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A Field Study of the Epidemiology and Clinical Manifestations of Parasitic Bronchitis in Adult Cattle

BY

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Introduction

WHETHER the incidence of husk in adult cattle has increased in recent years is difficult to determine. The incidence of a disease will appear to increase when it begins to be correctly diagnosed. Again, reports and discussions will only be forthcoming when interest is stimulated and this it may be by causes other than a marked increase in incidence. While husk in calves is, on a national scale, of far greater economic significance, the recent interest in the disease in adult stock has presumably been due to the circumstance that the cost of a single outbreak in the milking herd may be very great. In the case of one of the outbreaks to be described, outbreak L, a conservative estimate of the cost could be made. Only one of the 35 cows died but the cost, which came chiefly from the loss of milk and the need for extra feed, amounted to not less than £2,500.

The purpose of the investigation to be described was to determine whether any factors were common to all outbreaks and to provide a basis for laboratory experimentation. Now the smaller the number of outbreaks studied the more factors will tend to be common to them all and it is therefore necessary to study a large number. The 17 outbreaks to be discussed are selected from a larger number to illustrate the great diversity encountered.

Epidemiological investigation after the event has this shortcoming—that it can yield only circumstantial evidence, and most combinations of circumstances yield to more than one interpretation. The simultaneous consideration of several outbreaks, however, will give indications especially if some common factor or some uniformity of pattern emerges.

The difficulty of obtaining epidemiological information is aggravated, in the case of husk in adult cattle, by the spectacular and distressing course of the disease and the effect of this on those concerned. In the present investigation there was no recorded knowledge on which to base a previously designed questionnaire nor was it easy to recognise which factors were relevant. It was necessary therefore to obtain as complete a picture as possible and most of our informants showed surprising patience.

We very soon came to concentrate our attention on the grazing history and at first obtained information regarding this by a complex process of cross-examination. This procedure was very cumbersome and after a time a method of recording histories diagrammatically was devised. If such a diagram was constructed as the information was collected, contradictions and inconsistencies could immediately be spotted and clarified.

The Diagrammatic Representation of Grazing Histories

In the diagrams, of which 16 are reproduced in this paper, the position of the various groups of animals at different times through the grazing season is indicated. Each group of cattle is represented by a distinctive horizontal line, the vertical position of which indicates the physical position of the animals, the different enclosures being marked on a vertical scale. The position of the animals in time is given by the position of the line with reference to a horizontal time scale. In other words the position of different groups of animals is plotted with reference to enclosures (ordinates) and time (abscissae). Where a bunch of cattle is shown as being in more than one enclosure at the same time, then this means, either that they had access to one by night and the other by day, or that several enclosures were confluent and the animals ran in several, or again that the animals "lay back" on one and were allowed on to another for limited periods each day. Strip grazing appears in the diagrams as a series of steps where a back fence is used. The onset of symptoms is indicated by a vertical zig-zag line.

Discussion of Outbreaks

Before the present investigation was begun, a number of lungs from outbreaks had been subjected to helminthological examination. The findings have been briefly described elsewhere (Taylor & Michel, 1951). Immature worms were often present, sometimes in large numbers. In some cases only immature forms were recovered, in others, all stages from the early fifth stage to the mature adult were present. Since we were able to show that the development of the worms was often inhibited in the resistant host, their stage of development gives but little indication of when they were picked up. Furthermore, the bearing of this phenomenon upon the diagnosis of the disease by the usual methods is important and it has become our practice to examine a large number of faeces samples by a flotation method sensitive to the extent of one larva per 10 grammes of faeces and to base a positive diagnosis on the presence of even a single larva in a single sample.

At the outset of the present investigation it was known, therefore, that outbreaks in adults were not necessarily associated with the presence of large numbers of adult worms and for this reason it appeared unlikely that an outbreak was the consequence of a progressive build-up of infection on the pastures and in the cows. The adult bovine normally possesses an effective resistance to lungworms and if the build-up theory is untenable then it follows either that husk in adults is the outcome of sudden massive infection or that some unknown factor reduces the resistance of the host or increases its sensitivity.

It will be seen that in most of the outbreaks to be described and in a number recorded by other authors the onset of symptoms was sudden and occurred almost simultaneously in a whole group of animals. From this circumstance it seems to follow that there must have been some factor acting on all the affected cattle at some definite point in time. This might be either sudden exposure to infection or else the intervention of some other factor which precipitates the onset of symptoms. In both cases, in view of the degree of synchronisation one must look for some event affecting all the animals and preceding the onset of symptoms by a short rather than a long space of time.

Another problem concerned the ultimate source of the infection which might either have been introduced from outside or have always been present on the farm.

Outbreak A

Farm under same management 18 years. Beef rearing. All cattle sold off fat. Only new-born calves introduced, suckled on 22 home-bred nurse cows. Calves in boxes and cows brought in to them twice a day. All animals in show condition at all times. No previous history of husk and the disease said to be very rare in the district.

Grazing history shown in Fig. 1. A, indifferent old pasture. B, small paddock adjacent to a half-covered yard. Animals in yard generally given access to this paddock. C, excellent old pasture. D, highly productive lucerne and cocksfoot ley. Excess of grass in June such that moves on June 25th undertaken.

The groups of animals shown in Fig. 1 were : Group 1, 15 steers. Group 2, 17 steers not out before. Group 3, 28 stores not out before. Group 4, 7 young calves. Cows : 22 nurse cows. Devon steers : 40 two-year-old Devon steers which were afforded layerage for a short time in October.

Coughing first heard in A middle of June but one animal from Group 2 died June 2nd and found to be infected with lungworms. Group 2 also coughing slightly middle of June. One animal of Group 3 died suddenly middle of June, another August 2nd. Group 3 coughed hard and persistently, but did not lose much condition. A few showed signs of dyspnoea during August. Cows began to cough and lose condition middle of July. First died July 27th after short illness. Practitioner diagnosed fog fever. Another cow died August 1st and two more slaughtered August 2nd. Symptoms similar to first. Continued loss of condition in remainder, five more slaughtered. Groups 1 and 2 coughed only slightly and some not at all. Not possible to distinguish at this time which were Group 1 and which Group 2. No loss of condition. Group 4 severely affected. Coughing and dyspnoea began middle July, calves yarded but continued to deteriorate, 11 died between July 27th and August 10th. Faeces samples taken September 3rd : six nursing calves of Group 4 passing per 10 grammes of faeces, 759, 622, 47, 18, 9 and 0 larvae respectively. All other groups negative except one animal out of six of Group 3 with four larvae per 10 grammes.

Outbreak A represents the only case reported in this paper, in which the infection appears to have been introduced on to a clean farm. Every animal on the farm appears to have been susceptible but the variation in the severity of symptoms seems to depend upon the magnitude of the infestation to which they were first

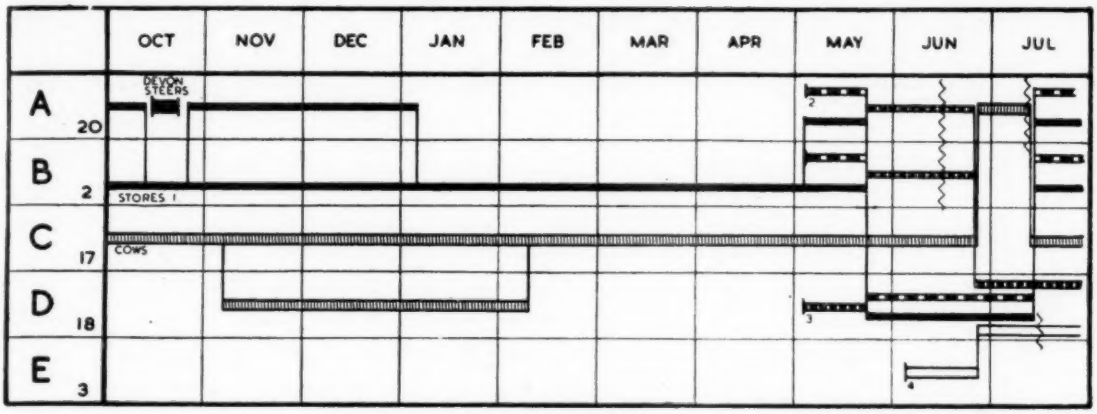


FIG. 1.—Outbreak A.

exposed. We would postulate that the Devon steers were the source of infection. The stores of Group 1 became lightly infected and kept the infection going on B, perhaps augmenting it a little. Group 2 also became lightly infected. They showed only slight symptoms which at first escaped notice until one died and was found to be infected with lungworms. Group 3 became more heavily infected. Not only were the symptoms more severe but two animals died. Group 3 appears to have become a rich source of infection, as may be seen from the catastrophic effect on the cows when they followed Group 3 in A. Similarly the calves of Group 4, when they came into D, bear testimony to the level of infestation that had been achieved by the combined effects of Groups 1, 2 and 3.

The diagram shows that Group 2 would not have had time to influence the level of infestation on the herbage of A and B, since they were here for less even than the minimum prepatent period of *D. viviparus*. On the other hand they must have contributed very largely to the infestation on D. It is a little difficult, therefore, to see why the symptoms in Group 3 were more severe than in Group 2 unless we postulate that the larvae disseminated by Group 1 were more effective in raising the infestation on the herbage in May than in April. Experimental evidence of the likelihood of this has since been obtained (Michel & Parfitt, 1955).

Outbreak B

Closed herd of Guernseys moved on to a fresh farm. No history of clinical husk in the herd but the disease had been of frequent occurrence on the new farm.

Grazing history in Fig. 2. All the grassland indifferent old pasture.

The nine calves which were in E all the winter were noticed to be coughing in April and lungworm larvae were demonstrated in their faeces. A little before the middle of June all 15 cows were noticed to be coughing. Three showed dyspnoea and a great fall in milk yield by June 25th. At this time four of the group of six calves developed pulmonary oedema and emphysema and two died. The lungs of one examined and contained a small number of half-grown worms. Faeces samples taken July 1st, all four survivors of six calves negative. One of the nine calves, three larvae per 10 grammes. Of 14 cows only the most seriously affected positive with 35 larvae per 10 grammes.

In outbreak B we see a similar case in which one group built up the infestation for the next. The nine calves evidently became lightly infected in E, the infection having been left behind by the cattle of the previous occupier. It is certainly a little difficult to understand why no symptoms were detected until April, but the important fact is that these nine calves were demonstrated to be passing larvae in April. The cows and the six calves picked up the infection so built up on E with painfully obvious results. One point of some interest, to which there will be occasion to return later, is the appearance of fog fever-like symptoms among the six calves shortly after they had been moved to an aftermath.

Outbreak C

Sixty head shorthorns with a sprinkling of Jerseys. Farm overstocked. Grassland unproductive as early growing grasses systematically punished. In-calf heifers in an enclosure at some distance from rest of farm. Younger heifers, dry cows and calves with milking herd. Herd taken over by present owner eight years previously together with farm. Husk common before this in calves and an outbreak in the present owner's first year. No clinical husk since.

Grazing history is shown in Fig. 3. Access to E, B and D is through A. Access to A through F. C reached through D. A, moderately good old rye grass and cocksfoot pasture. B, a ley in its seventh year. Sown species disappeared—now mediocre Agrostis rye grass pasture but with fair proportion white clover. C, a predominantly grassy ley of which half was three and half four years old. D, similar to B, but with little clover, half acre by gateway became rank and unpalatable. E, sown under oats in previous year. F, the collecting ground, a muddy half acre growing nettles, weeds and a little rank grass. G, old pasture at some distance from rest of farm.

"Cows" numbered 25, in-milk and dry. "In-calf heifers" numbered 10. One was drafted into the milking herd on July 25th. Four on September 3rd. "Bulling heifers," 12, one and two years old.

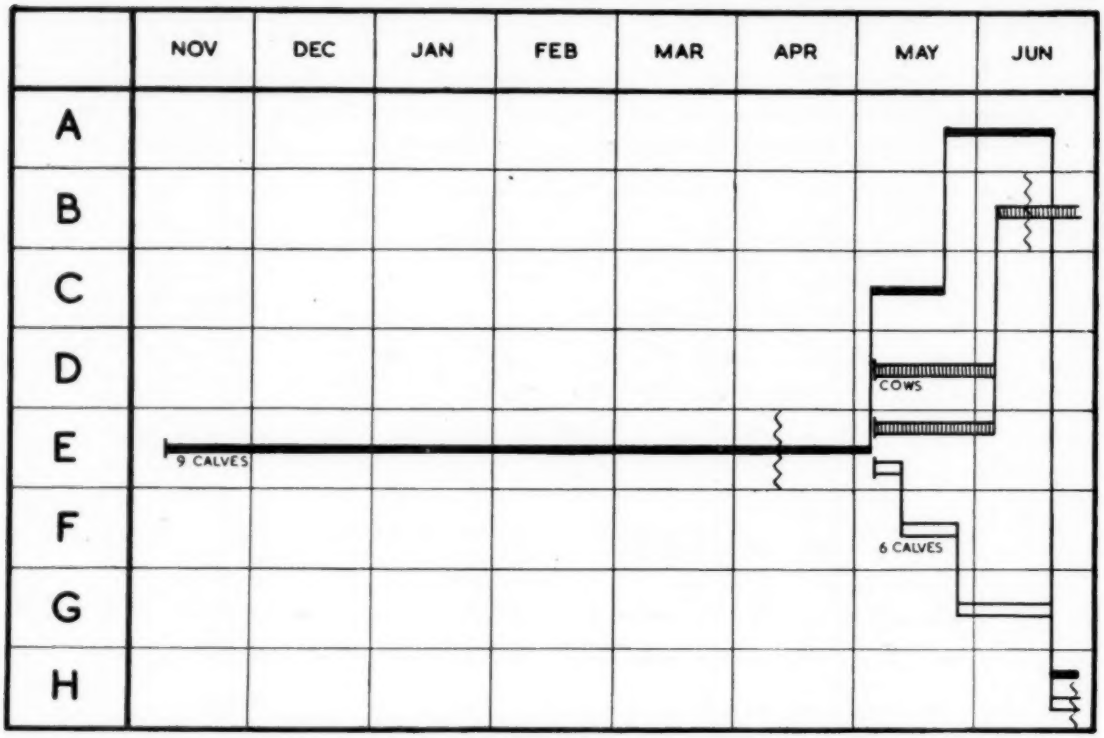


FIG. 2.—Outbreak B.

"Calves," eight heifer calves between five months and one year.

Onset of symptoms difficult to determine as owner had been away and labour difficulties in cowshed. First noticed middle of August and indications were that cows began to cough only a few days before this. Only cows and calves affected and symptoms not severe. No noticeable loss of milk. Only two animals lost condition. These were newly calved heifers from G. These two the most severely affected and occasionally dyspnoeic as were a few other cows. Young heifers entirely unaffected.

Outbreak C represents a situation in which the infection had clearly been constantly present on the farm and in the herd in a sub-clinical form. The parasite was known to be present eight years before and no animals had been introduced for some years. Two facts appear to be particularly pertinent. The heifers were not affected, and this was the first year in which the cows were separated for a time from the young stock. Either the cows or the bulling heifers must have acted as the source of infection. The calves would pick up this moderate infection as soon as they came out and by July 28th will have begun to raise the level of infestation on the ground. The cows returning to this, and suddenly exposed to it, became clinically affected. It might be argued that the bulling heifers remained unaffected either because they were more resistant or because they were exposed more gradually to the higher level of infestation brought about by the calves than the cows. This latter explanation is unacceptable as will be seen from the diagram. The calves would not have had any effect on the level of infestation on the herbage until approximately July 28th, the day the cows returned. The fact that the bulling heifers were resistant to infection might be taken to suggest that they had been more recently infected and were thus more likely to have been the original source of infection.

Outbreak D

Attested shorthorns, herd closed seven years. No clinical husk since outbreak in yearlings 12 years before.

Grazing history shown in Fig. 4. A, excellent old pasture dressed in spring with 3 cwt. per acre of a complete fertiliser. Divided into three portions until end of June. B, three-year-old ley Irish perennial rye grass, timothy, S.100. C, three years old, consisted of little but meadow fescue which began to grow late but then grew with great vigour. D, rich old pasture near the buildings. Half the cows were wintered here and it became badly poached. E, a small paddock, once an orchard, grazed in previous autumn by cattle now 18 to 22 months old and subsequently and until Christmas by odd animals. F, another small orchard. G, a variety of enclosures details of which could not be secured. Cows were 36 head in-milk and dry. Heifers : 20 head between 18 and 22 months had been out in previous summer. Calves : between six months and one year. Neither the heifers in June nor the cows later could cope with the growth of grass in C. When cows taken out it was still possible to mow it and secure a ton of hay per acre.

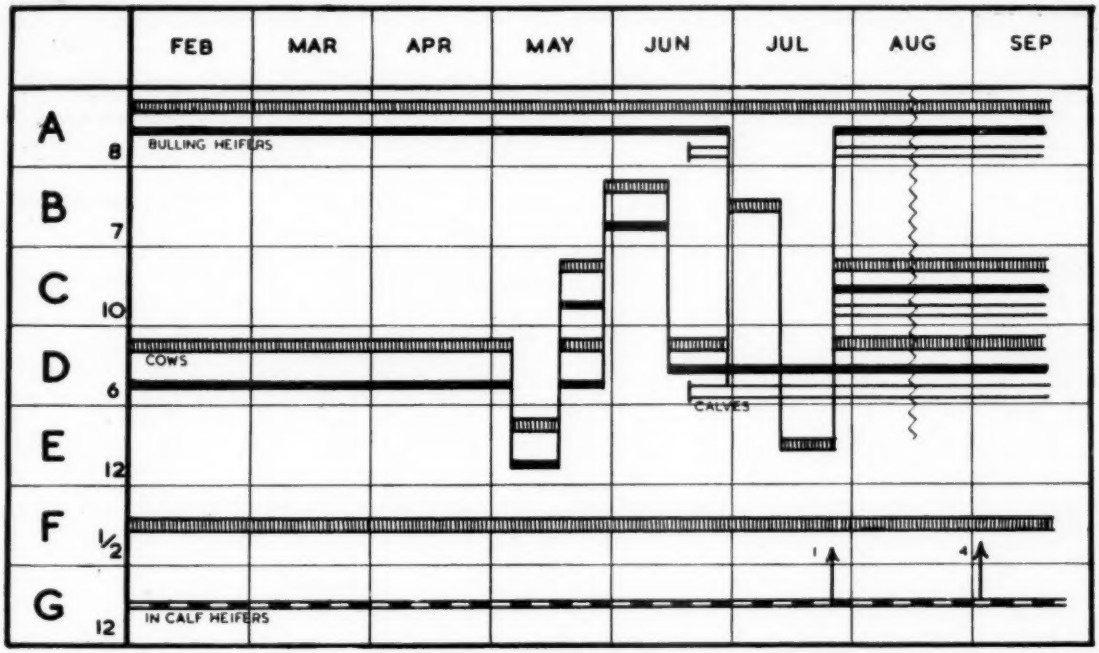


FIG. 3.—Outbreak C.

Owner noticed loss of appetite in cows beginning approximately June 24th. Symptoms appeared July 7th. Coughing, dyspnoea and expiratory grunt in some animals. Practitioner diagnosed "protein allergy"—faeces negative for Dictyocaulus larvae. Sharp fall in milk yields from 75 gallons per day to 48. First calf heifers worst affected. Faeces samples taken in August positive. First cow to show symptoms was one that had been housed until late June. Heifers in C for one week beginning June, then moved to entirely separate grazings, G in diagram. A little coughing heard among them during August. Returned to B and C end of August and a fortnight later were coughing violently. Young calves from F were moved to E after cows taken out. Fortnight later owner suspected one or two were coughing and they were housed again.

In outbreak D the cows went off their feed and appeared "off colour" when they went into C and E. They showed symptoms of husk 12 days later. It will be seen from the diagram that the heifers had been in C a fortnight before the cows and it may be justifiable to postulate that they were the source of infection. According to this view the conditions in C must have been very favourable for the development of the larvae and in so far as it was still possible to mow the field when the cows were removed and obtain a ton of hay to the acre, we have presumptive evidence that an excess of keep may favour the larvae. It will further be seen that the heifers returned to C in August and began to cough violently a fortnight later. We may postulate, therefore, that though a cut of hay had been taken the aftermath was still heavily infected. This interpretation of the outbreak leads to some difficulty. If the cows became infected in C then their infection would not have been patent by the time they left E. The mild cough that was seen in the calves 14 days after they came to E is therefore not easy to explain although three possibilities can be suggested. The owner, who by this time was highly alarmed at the course events were taking, may have imagined he heard coughing, the calves might have picked up the infection in F, and it was not possible to determine conclusively what animals had been here before the calves, and lastly, there could have been the odd carrier among the cows.

Outbreak E

Small mixed herd closed five years. No symptoms suggestive of husk seen during this time. Poor undrained clay soil, many springs and standard of husbandry very low. Ten cows, three heifers of which one in calf.

Grazing history in Fig. 5. Heifers in A by day and in C by night. Until end of March cows also in A by day and housed at night. Thereafter in A by night and other pastures, as indicated, by day. A, sloping, badly poached but rich in wild white clover in parts. B, a one-year ley in its third year. Sown species had died and bare earth only partly colonised by indigenous species, fair amount wild white clover. C, moderate Agrostis, rye-grass pasture with many springs. D, under water till mid-March. Herbage coarse and rank, no clover. E, a freshly dunged stubble.

In-calf heifer developed persistent cough about June 10th to July 5th, other heifers and cows began to cough, and with increasing intensity. July 19th the only Guernsey had high temperature, emphysematous lungs and fall in milk yield. Few days later the only Jersey similarly affected. Both recovered within a fortnight.

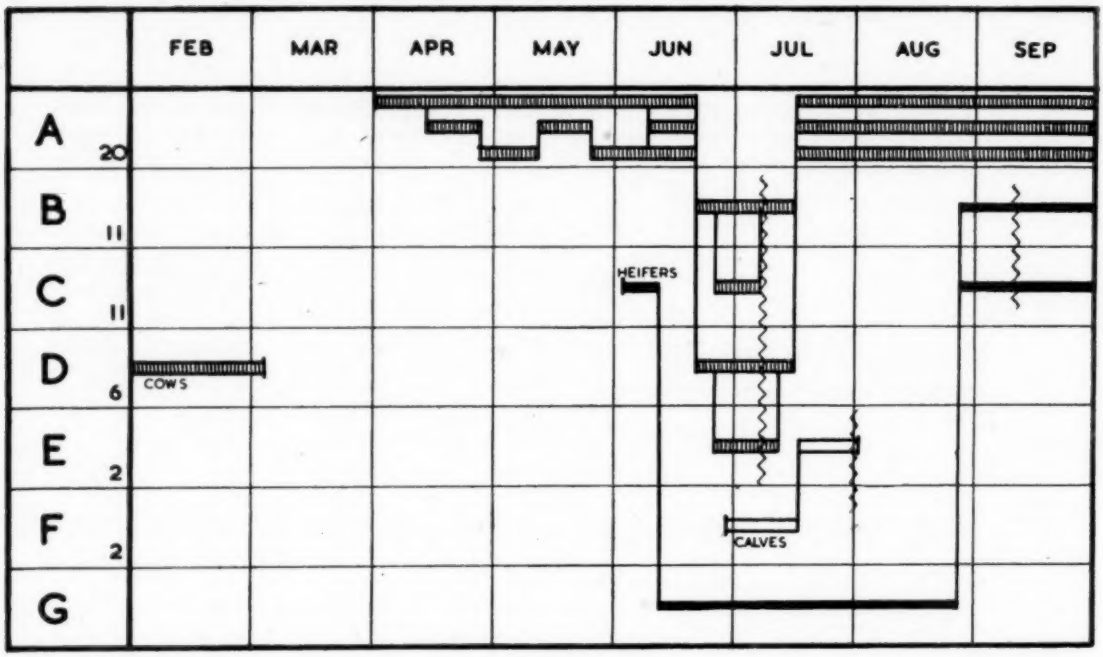


FIG. 4.—Outbreak D.

Faeces samples taken July 19th all negative save in-calf heifer with one larva per 10 grammes, and another heifer with two larvae per 10 grammes.

In outbreak E it is reasonable to postulate that the in-calf heifer represented the immediate source of the infection that was picked up by the cows. How, and in what manner, the in-calf heifer came to be infected is more difficult. It would appear, however, that she could not have picked up the infection during the spring as the grazing of the cows and heifers overlapped and had there been a dangerous level of infestation on the ground other animals would presumably have shown symptoms at the same time.

One is left, therefore, with the possibility that the in-calf heifer was carrying immature worms, inhibited in their development, which resumed their development, grew to maturity and in doing so produced symptoms.

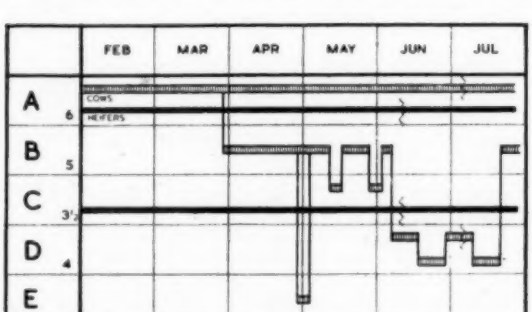


FIG. 5.—Outbreak E.

Outbreak F

Old-established closed herd attested Jerseys. Milking cows and young stock always used entirely separate grazings. Clinical husk never seen. Milking cows, 25 head. Heifers, 10 in-calf, 17 bulling. Calves, five head, three to four months. Grazing history in Fig. 6. All moderate old pasture save A, a newly sown ley not grazed before.

Amount of coughing among cows attracted attention May 22nd, though two newly calved heifers coughing for a fortnight before this. Condition continued to deteriorate and coughing more evident and after three weeks marked loss of weight and drop in milk yield. June 29th (vertical dotted line in diagram), one cow acutely dyspnoeic, pulmonary emphysema. By second week July most of other cows similarly affected.

Exact time when heifers in P not known but the fact they were there before the calves established. Calves first noticed coughing August 4th and one that died mid-August harbouring large number lungworms. Heifers never coughed.

In outbreak F symptoms began 28 days after the cows came out to grass. If we may assume that significant infestations of lungworm larvae do not survive over winter on the ground, and all the evidence supports this view, then it follows that one or more of the cows must have been a source of infection. There is no possibility that there was a build-up of infection as there just was not time.

The time interval of 17 days between the entry of animals to the ley is interesting. If, as must have been the case, there was a source of infection among the cows at the time they entered A then infective larvae would

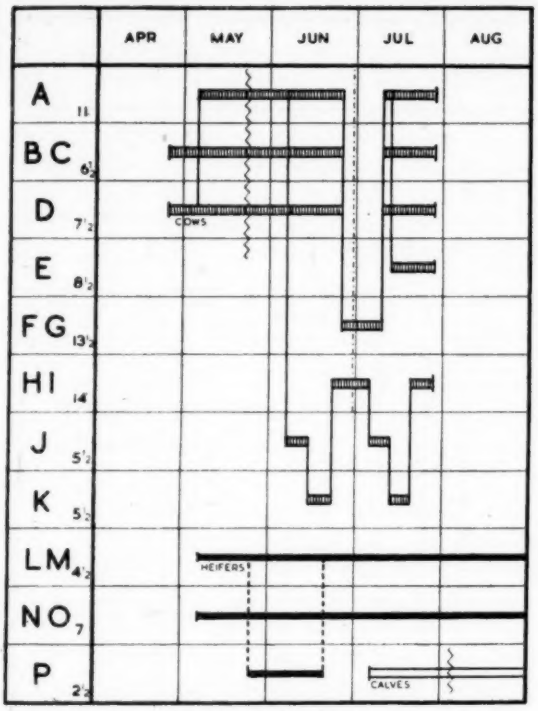


FIG. 6.—Outbreak F.

have been available on A between five and seven days later and the symptoms appeared 10 to 12 days after this, the incubation period most commonly encountered in this investigation as well as experimentally when animals that have had previous experience of infection are re-infected. The suggestion is therefore that the ley A represented a factor particularly favourable to the development, survival or availability of the larvae.

That the infection was always present in the young stock side of the farm emerges from the circumstance that the heifers were resistant and acted as a source of infection on P.

It would appear, therefore, that the infection was brought from the young stock side to the adult side and suspicion must fall on the two newly calved heifers, which began to cough a fortnight before the rest of the milking herd and which were among the least severely affected.

Outbreak G

Guernsey herd closed some years. No history of clinical husk in previous years. Eighteen cows. Eight heifers which had been out in previous year, five heifers not out before. Grazing history in Fig. 7. Grassland consisted either of old pastures or very elderly leys and presented no noteworthy characteristics. B was very bare all the season. C was fairly lush in May but cows left stalks only and there had been no bottom growth when they left. Cows in G by night throughout the season.

About seven cows coughed mildly in June but attracted little attention. July 4th heifers began to cough, four of five not out previously showed acute dyspnoea and great loss of weight. Eight out in previous year developed a mild cough but were not otherwise affected. End July severely affected heifers had begun to improve but still coughed. The four calves remained unaffected.

In outbreak G the simplest explanation is that the source of infection was among the cows if only because the heifers followed the cows, while the cows never followed the heifers. It seems difficult to understand why the appearance of symptoms among the heifers was so long delayed, especially in view of the fact that their onset was so sudden and their occurrence was simultaneous in all the animals affected. The cows began to cough 12 days after going into F and if we postulate that they picked up the effective dose of larvae here then it is necessary to explain why this did not happen in C or E. At the time the outbreak was visited it was not possible to discern in what manner F differed from the other pastures, but there can be no doubt that there was more grass in F when the cows were here the first time than in C or E and certainly it was growing more rapidly at that time. Again, our earlier supposition that the larvae are more effective in raising the level of infestation on the ground in May than they are in April would offer an explanation.

Outbreak H

Ayrshires on farm 16 years and no female stock introduced during that time. No stock of any kind introduced during past four years. Management, exactly the same every year, revolved round use of two irrigated water meadows I and J in Fig. 8. In year after herd brought to this farm there had been an outbreak of husk in young stock grazing on these meadows and thereafter they were used for milking herd and young stock grazed high-lying pastures on chalk. No clinical husk had been seen since.

Grazing history shown in Fig. 8. Fifty cows and followers in three groups : in-calf heifers, stirks first out in March and young calves. Cows always on old pastures B and C by night and grazed water meadows and a clover ley in strict rotation. Water meadows irrigated as soon as cows taken off. All grassland received 4 cwt. per acre complete fertiliser per annum. Dry cows with milking herd but paddock G for odd cows to be kept under observation, etc. During winter a group of dry cows had run separately in E. Symptoms not serious though onset sudden. Time of onset in each group indicated in Fig. 8 by vertical zig-zag line.

Outbreak H suggests that in this case an appreciable proportion of the cows were acting as a source of infection for a considerable period. The dry cows in E clearly left infection here, as may be seen from the effect on the stirks which had not been out in the previous year.

Similarly the cows left A infected and in spite of the intervening hay cut, the in-calf heifers showed symptoms in less than a fortnight. The calves remained free of infection while they were in F, but contracted the disease in G which had accommodated odd cows. It would appear that the infection had not persisted for the five months in which F lay empty. From the circumstances of this outbreak, it would appear, therefore, that the larvae can survive for three months, but

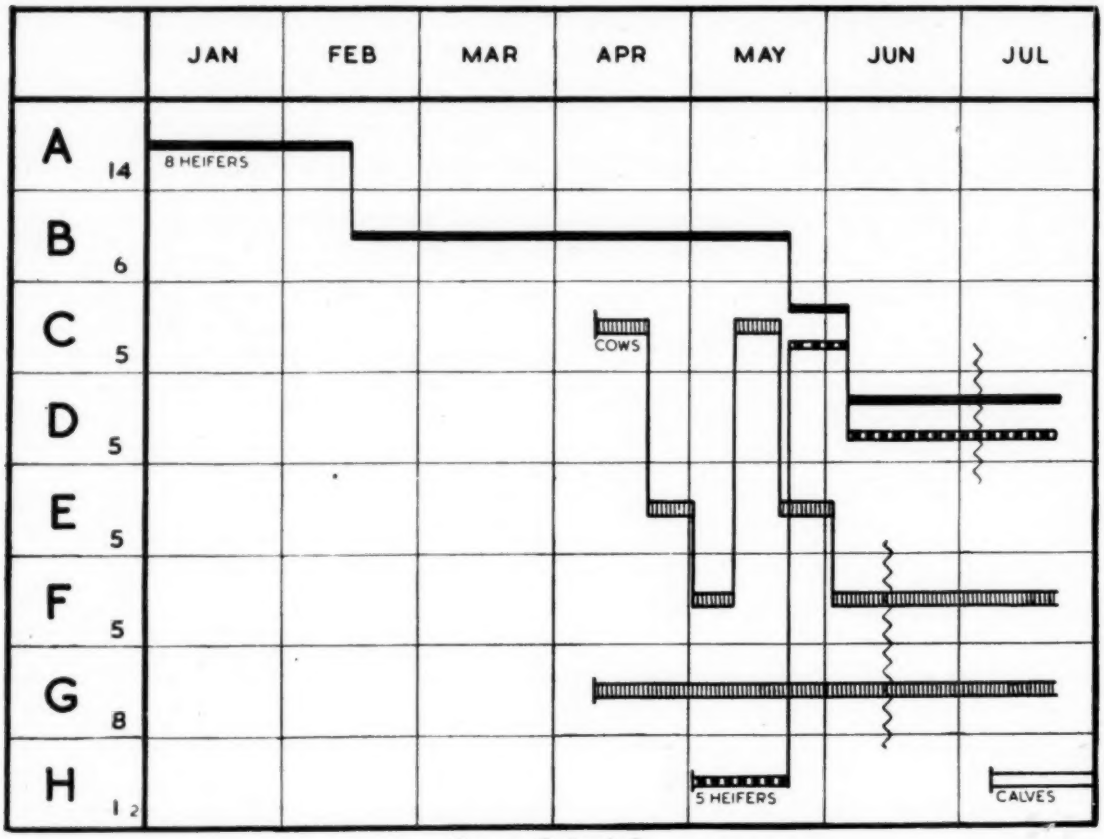


FIG. 7.—Outbreak G.

not for five, a conclusion that corresponds well with the results of laboratory observations on this subject.

The appearance of symptoms in the cows is of interest. It will be seen from the diagram that they appeared 14 days after the cows returned to J. We may postulate, therefore, that there was something about J which was particularly favourable to the lungworm larvae. It would appear that the larvae were more successful at developing to the infective stage and finding their way back to the cows under the conditions of a water meadow than on higher-lying and drier grazings.

Outbreak I

Infertile holding on Weald clay. In hands of present owner four years. Thirty-five Ayrshires and their followers. Grazing of leys limited by time, cattle "lying back" on old turf. This was the first year that grass had been available throughout the season. Normally dependent on rape in August and September.

History shown in Fig. 9. A, directly reseeded. First grazed in August. B and C sub-divisions of same old pasture, matted turf of Agrostis and Yorkshire fog. D, S.100 cocksfoot ley part two years part three years old but considerably deteriorated. E, directly sown in previous year with an S.100 ley mixture. Had been overgrazed by rabbits and grasses killed. Now only S.100 and indigenous wild white clover. Very highly productive. F, indifferent old wild white clover rye-grass Agrostis pasture. G, sown under wheat in previous year with hay loft sweepings. Almost entirely Yorkshire fog which stock found unpalatable.

Onset of symptoms sudden but not very serious. Only cows affected and oldest cows and second calves the worst and their milk yield reduced. Faeces samples taken October 15th—two of five had 11 and 9 larvae per 10 grammes respectively.

In outbreak I we see another case where symptoms appear 12 days after the cows return to a lush clover ley. In this case the time-grazed ley E would seem to have provided conditions favourable to the lungworm larvae.

That the bulling heifers were not a source of infection may be seen from the fact that the calves in C remained unaffected. At the same time the cows cannot have left an appreciable level of infestation in D as the bulling heifers remained unaffected.

Outbreak J

Guernsey herd managed in same way 28 years. Sixty to 65 milking. Ten heifers drafted into milking herd each year. Young stock reared on adjacent farm. Grazing of cows and young stock hardly ever overlapped. A little coughing among calves every year but no symptoms among cows since an outbreak of undiagnosed

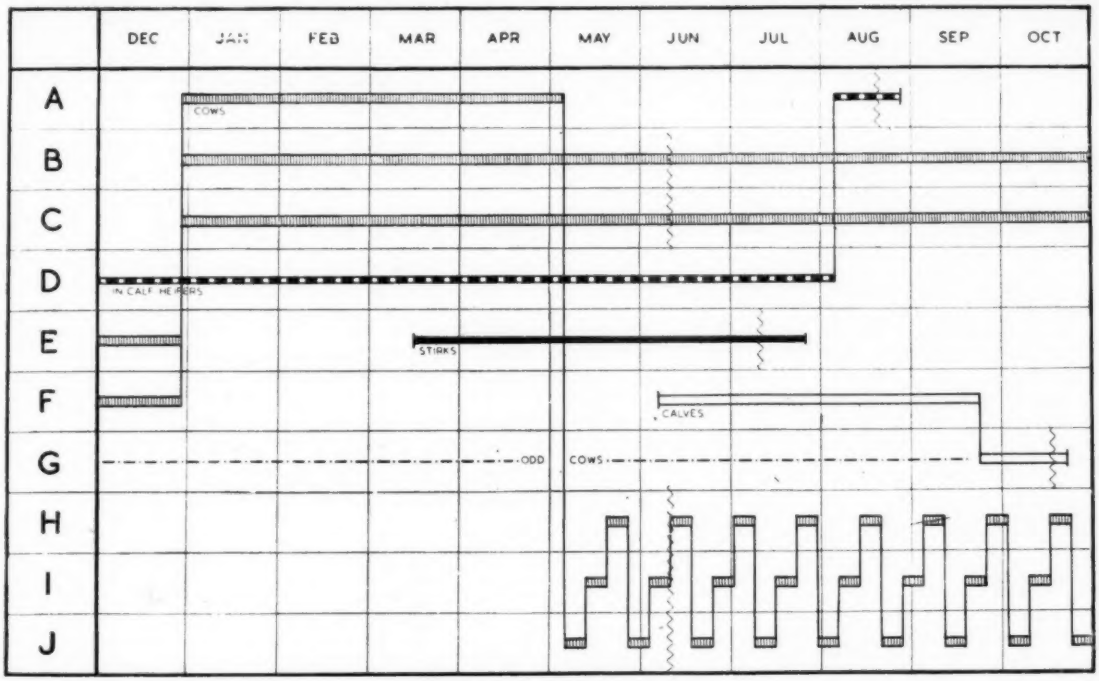


FIG. 8.—Outbreak H.

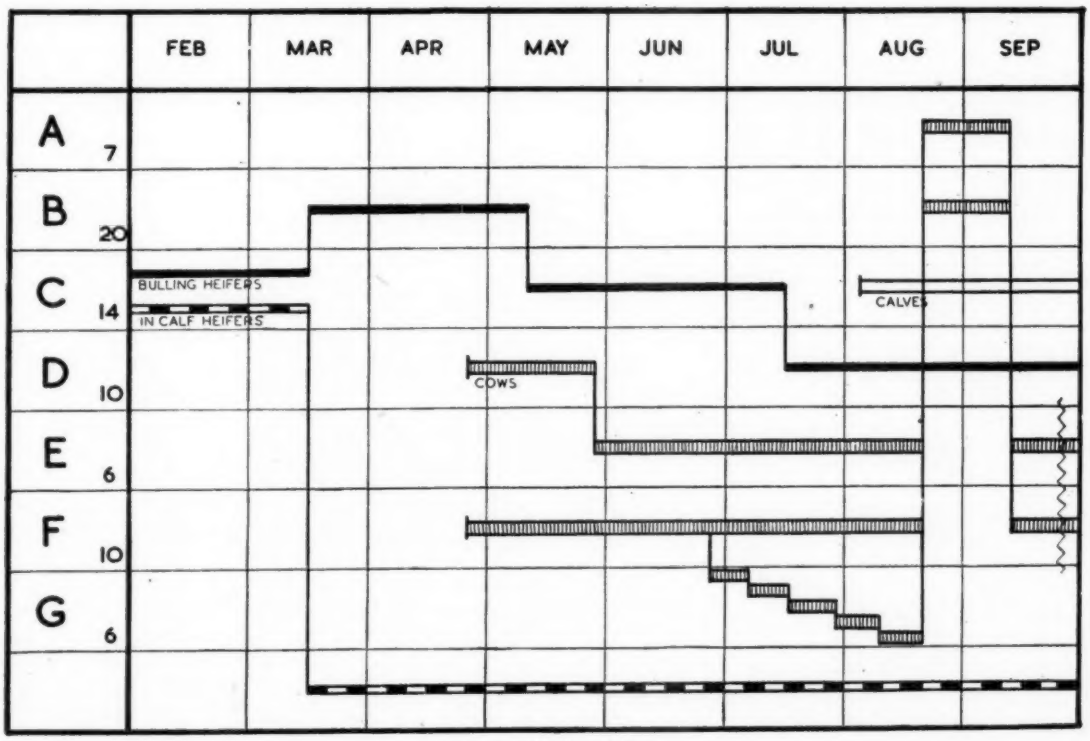


FIG. 9.—Outbreak I.

respiratory disease some 20 years before which practitioner now thought had been husk.

Cow grazings (Fig. 10) 35 acres of improved old pasture. Originally this served as exercise ground but extent of improvement such that they now supported herd throughout grazing season. Four to six cwt. complete fertiliser given yearly. Access to C through B and since no water in C, C could only be grazed together with B. Growth of grass very great in this year and second circuit around enclosures took twice as long as first as the cattle found it difficult to master the growth. Partly for this reason that C missed out. When cows ultimately returned to C there were very large quantities of grass indeed.

At this time (July 22nd) bailiff noticed loss of appetite and cows "off colour." Twelve days after return to C first symptoms appeared. Coughing increasing in intensity over three days when one animal dyspnoeic, remainder coughing violently. Within a few days 10 to 12 emphysematous and dyspnoeic. Considerable fall in milk yields. Beginning of September coughing still violent and six cows still with dyspnoea. Faeces samples were taken from various age groups. Three of 12 samples from 9- to 18-month-old heifers had 153, 120 and 1 larvae per 10 grammes. Twelve samples from two-year-old heifer were negative. Eight of 24 samples from milking cows had 802, 259, 77, 20, 5, 3, 2 and 2 larvae per 10 grammes respectively. Similar samples taken in October and monthly thereafter for six months were all negative.

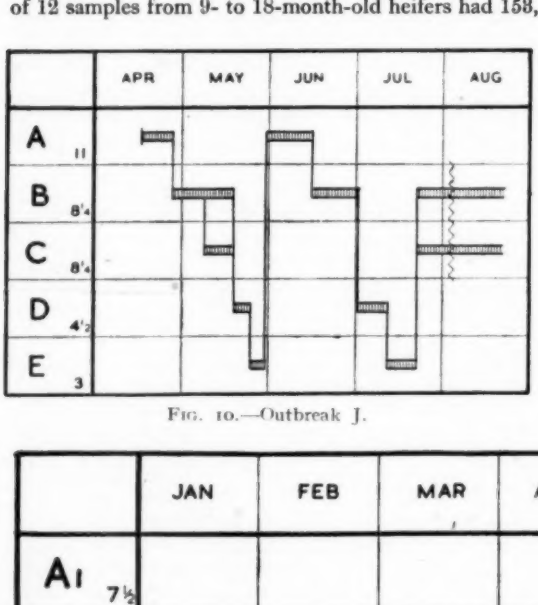


FIG. 10.—Outbreak J.

In outbreak J again the symptoms appear 12 days after the cows returned to an enclosure in which there was an excessive amount of keep. Here again, as in outbreak F, the infection appears to have been present in the young stock every year and some accident of fortune conspired to bring it from one part of the farm to the other and at the same time to provide suitable conditions for its multiplication. The loss of appetite reported by the bailiff though it is not necessarily the most reliable form of evidence gains strength from the similar observations made in outbreak D and suggests that a massive infection was being picked up in C.

Outbreak K

Closed herd attested Guernseys moved two years before to a poor clay farm. No history of clinical husk in herd but outbreaks had occurred on the new farm before it was taken over.

Grazing history in Fig. 11. A, rye-grass and S.100 and S.123 ley in its first year. Dressed in January with 3 cwt. per acre complete fertiliser. Highly productive. Divided in half and part mown. B, home paddock indifferent herbage but much wild white clover. C, a series of contiguous meadows by a stream, defective drainage, herbage of the poorest quality.

"Cows" in diagram 24, dry and in milk, of which five were first-calf heifers. "Young stock" were 15 heifers 9 to 24 months, one barren cow and one young bull. The younger heifers had not been out in previous season. All had been yarded during winter. Cows were put on B two hours daily for exercise during winter.

July 13th, one of first-calf heifers noticed apart from herd with head down, mouth open, tongue protruded, shallow rapid panting and periods in which each expiratory effort accompanied by a loud grunt or groan. Temperature 105° F. Practitioner diagnosed "fog

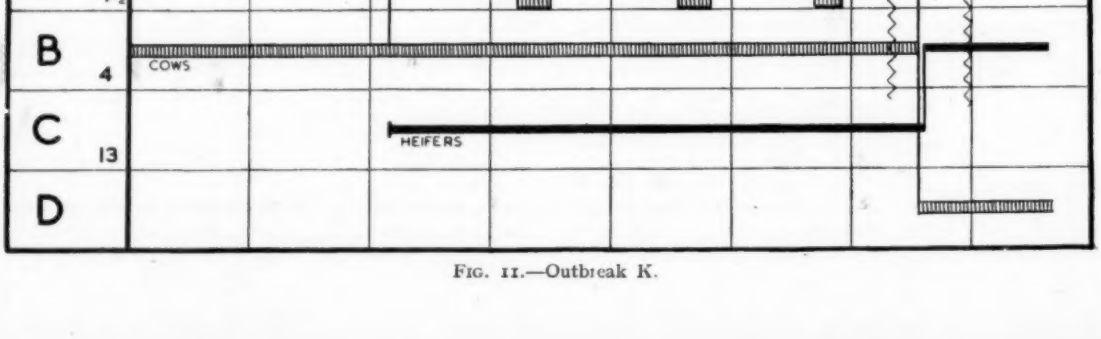


FIG. 11.—Outbreak K.

fever." Within five days four more animals, two of which were first-calf heifers, showing same symptoms. Second animal to fall ill died July 22nd. The first animal to fall ill brought to the laboratory as beyond hope of recovery and slaughtered on July 25th. Much improved on arrival and lungs had only a few patches of emphysema. Three thousand six hundred and sixty lungworms recovered ranging from earliest fifth stage to mature adult. About this time position was that six more animals seriously ill, of which three were first calvers, three second. All these had high temperatures and emphysema. Laboured rapid abdominal panting and ribs held out fixed. Intermittent coughing. Other members of herd mild symptoms of respiratory distress and much coughing. There had been sharp fall in milk yield and many animals had lost weight. Only bull who had been housed and one old cow that had grazed separately not affected. August 9th, another heifer (Fig. 17) brought to laboratory for slaughter. Emaciated and fog fever symptoms. Very weak. Lungs grossly oedematous and emphysematous and contained 680 worms of all sizes. August 17th, breathing of less severely affected animals no longer distressed but still much coughing. Nine animals still severely ill but no emphysema detectable. Had lost condition and none coughed much. Four animals had already died. Two more died with almost entirely consolidated lungs. A fortnight after young stock moved to B they began to cough. After a month some were beginning to be dyspnoeic.

In outbreak K the symptoms appeared suddenly 12 days after the cows were put on the aftermath A. In this case, however, we may be certain that the aftermath A was helminthologically clean. In view of the fact that the symptoms appeared almost simultaneously in all the cows affected, we must conclude that there was some precipitating cause and the move to aftermath would seem to be the only likely factor to have operated in this way. The result when the heifers were moved to B and the fact that the symptoms appeared in them in only 12 days indicates that the level of infestation on B was high. Only the cows can have given rise to this infestation.

Outbreak L

High yielding herd of Jerseys closed nine years. Standard of cowmanship very high but yields obtained on what would be considered inadequate diet. Young stock not done well. No history of husk since serious outbreak in adults eight years before. Usual practice to house all cattle Christmas to April or May.

Calves normally kept in during what would be their first grazing season but out this year in piece (A_1 in Fig. 12) fenced off from A. In June they were allowed to run on remainder of A on two occasions. A, poor old pasture on thin clay soil. B, three-year ley sown with Cockle park mixture. Thin soil on chalk but very productive owing to heavy dressings complete fertiliser. Divided in half and halves strip grazed alternatively, cows running back over grazed strips. C, small paddock near buildings accommodated odd cows. D, new ley almost entirely H_1 rye-grass. E, mainly S.23 and S.24 rye-grass and unproductive end of May and June. F, direct reseed part H_1, S.100 and S.123 part timothy and meadow fescue. Had come up dirty and weeds cut and carried before cows let in.

June 28th, herdsman detected slight cough in cows. Beginning July there was a hot spell and respiration of several cows became distressed and high temperatures. General condition and milk yield of remaining cows rapidly deteriorated. First moved to A and C which were closer, as movement aggravated symptoms. Later kept in altogether. Standard of nursing high so that only one cow died end of July and found by practitioner to harbour large numbers mature lungworms. Altogether 20 of the 35 cows seriously ill but first-calf heifers not worst affected. Dyspnoea, coughing and progressive loss of weight continued till mid-September. Onset of symptoms in young stock as indicated but not very severe.

Faeces samples from four cows July 26th contained 0, 1, 19 and 26 larvae per 10 grammes respectively. August 22nd, one of three calves had eight and one of three heifers 12 larvae per 10 grammes.

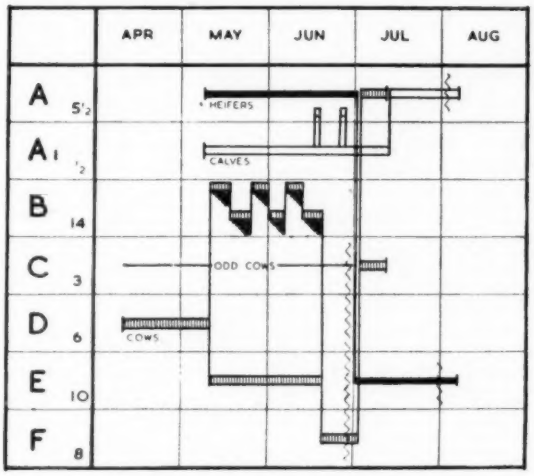


FIG. 12.—Outbreak L.

In outbreak L mild coughing was noticed by the unusually observant herdsman nine days after, and extreme symptoms appeared 12 days after the cows were moved to F, a recently cut direct reseeding. This also must have been clean, as it had carried no cattle for eight years. Again the cows are the only possible source of infection and, as may be seen from the effect on the heifers, were passing larvae on June 19th when they left E.

One is again left to conclude that F provided something to precipitate the appearance of symptoms.

Outbreak M

Herd of Guernseys, established five years. Outbreak of husk in young stock in first year. None since. In previous year cow brought in in July had been coughing hard by September. Lungworm larvae demonstrated in faeces and cow disposed of end of September in very emaciated condition.

All grassland elderly leys of indifferent merit but rich in wild white clover. "Cows" in Fig. 13 were 27 head,

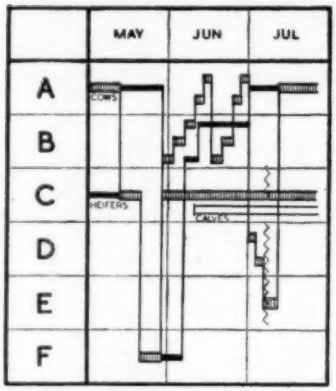


FIG. 13.—Outbreak M.

in-milk and dry. "Heifers" were 20 in-calf and bulling. "Calves" were five heifer calves, all under 12 months, out only a few hours daily.

July 7th, 10 cows began to cough and some deterioration in bodily condition and milk yields. No other animals showed any symptoms and outbreak subsided fairly quickly. November 4th, one of the heifers died suddenly with grossly oedematous and emphysematous lungs which, on thorough examination, revealed two microscopical lungworms.

In the case of outbreak M there can be little doubt that the disease was endemic on the farm. The susceptible cow brought on to the farm in the previous year would appear to offer adequate evidence of this. In the outbreak under consideration there is little means of knowing whether the source of infection was among the heifers or the cows, as not only did the heifers graze after the cows but the cows also followed the heifers. From the fact that the heifers remained unaffected we must conclude, therefore, that they possessed some resistance. The fact that the calves remained unaffected would seem to suggest that on the bare home paddock C the level of infestation remained insignificant while on the better covered A and B it was able to become meaningful. Alternatively, one might seek to deduce that the heifers were the source of infection and the cows, while they became infected in A or B, never passed any larvae in their faeces.

One might remark in passing that the symptoms appeared seven days after the cows were moved to a clean aftermath.

It will be recalled that in the case of outbreak B symptoms in the six calves appeared a week after they were put on a clean aftermath. It might likewise be argued that in outbreak F the extreme symptoms, indicated in the diagram by the vertical dotted line, appeared seven days after the animals were moved to the aftermaths H and I.

It would appear, therefore, that the sudden onset of symptoms in a whole group of animals may be associated with sudden exposure to infection or with a move to aftermath.

Outbreak N

Jerseys had just replaced Shorthorns on this farm. Three years previously there had been a husk outbreak in Shorthorn heifers. Two years before present outbreak, which was second in two consecutive years, process of replacing one breed by another began. All Shorthorns disposed of by following April but small number of Jerseys continued to come in until following winter.

Grazing history shown in Fig. 14 very complex. All grassland highly productive leys strip grazed with electric fence, cows being allowed to run back over the grazed strips. Different fields grazed by day and by night. Abundant grass throughout year. When cows returned to A_1 for third time June 18th, growth such that they were taken out again and field mown for hay. "Cows" in diagram were 50, milking and dry. "Heifers" were seven in-calf and 12 bulling heifers. These had been affected in previous year and continued to cough throughout winter and spring. Five calves were turned out May 14th and a further four June 4th. These four were brought in again because not thriving.

About July 8th, 35 to 40 cows began to cough. Some worse than others and slight loss of condition and milk. Little dyspnoea but two cows developed secondary

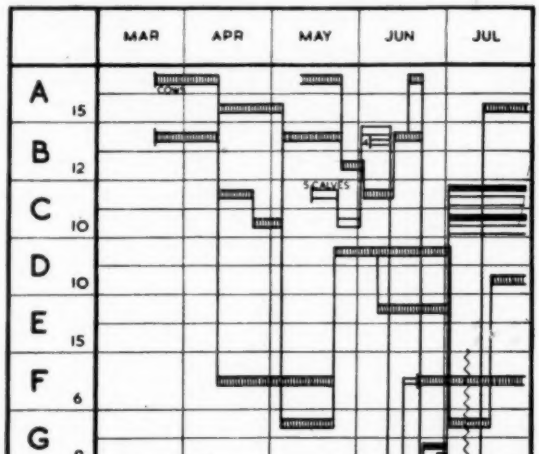


FIG. 14.—Outbreak N.

bacterial pneumonia. Similar proportion had been affected previous year but difficult to determine whether same animals unaffected both years. Fair number must have been affected in both outbreaks. Calves brought in again also coughed. Faeces samples taken August 2nd. Three out of eight heifers had 4, 9 and 12 larvae per 10 grammes, three of 23 cows had 1, 11 and 23 larvae per 10 grammes. Of three yarded calves two had 14 and 5 larvae per 10 grammes.

In outbreak N the calves cannot have built up the infection as they appear to have done in outbreak C as neither group had sufficient time to do so. On the other hand the situation in A where there was so much

grass that the animals were taken out and the field mown is strongly reminiscent of outbreak D, if indeed these are the conditions in which lungworm larvae flourish then the cows would have become infected here.

It would appear, then, in outbreak N that the cows were passing larvae and that particularly favourable conditions arose, perhaps in A, for the cows to take in a massive reinfection. What is particularly of interest in this case is that two outbreaks occurred in successive years. This we have seen on only one other occasion and in circumstances very similar to this in that one herd was being gradually replaced by another.

Outbreak O

Attested herd of Friesians with sprinkling of Jersey. Closed four years. No clinical husk on farm for at least 15 years.

Grazing history in Fig. 15. A, a second-year ley, rich in clover, had grown vigorously all the season and never been bare. B and C, permanent pastures of average quality, mown for hay in June. D, sown in previous year under corn and stubble grazed by cows. Lush when cows put in in April but bare when they left and remained so. E, an elderly ley mown for hay in June and dunged about July 24th. F, sown directly previous autumn. Strip grazed by cows and then shut up for hay. G and H, uninspiring old turf. J, sown direct in spring.

"Cows" were 27 head, in-milk and dry. "Heifers" were all the young stock above nine months old. Eight were in-calf, of which four Friesians and one Jersey were due October and November. These five, though running with the other heifers, had always kept apart from them.

September 18th, one or two cows noticed coughing slightly. Young stock inspected and found also to be coughing slightly. September 21st, five heavily in-calf heifers developed acute dyspnoea. Shallow abdominal panting, loud expiratory grunt. Lungs oedematous and emphysematous. One died September 23rd, another aborted September 28th. September 30th, these extreme symptoms showed no sign of abating. Younger heifers still coughed mildly. Lungs of animal that died contained 1,100 mature and 520 immature *D. viviparus*.

In outbreak O the five in-calf heifers developed extreme symptoms within hours of each other. It is certainly conceivable that they became infected in D, even though this made little growth and there was a

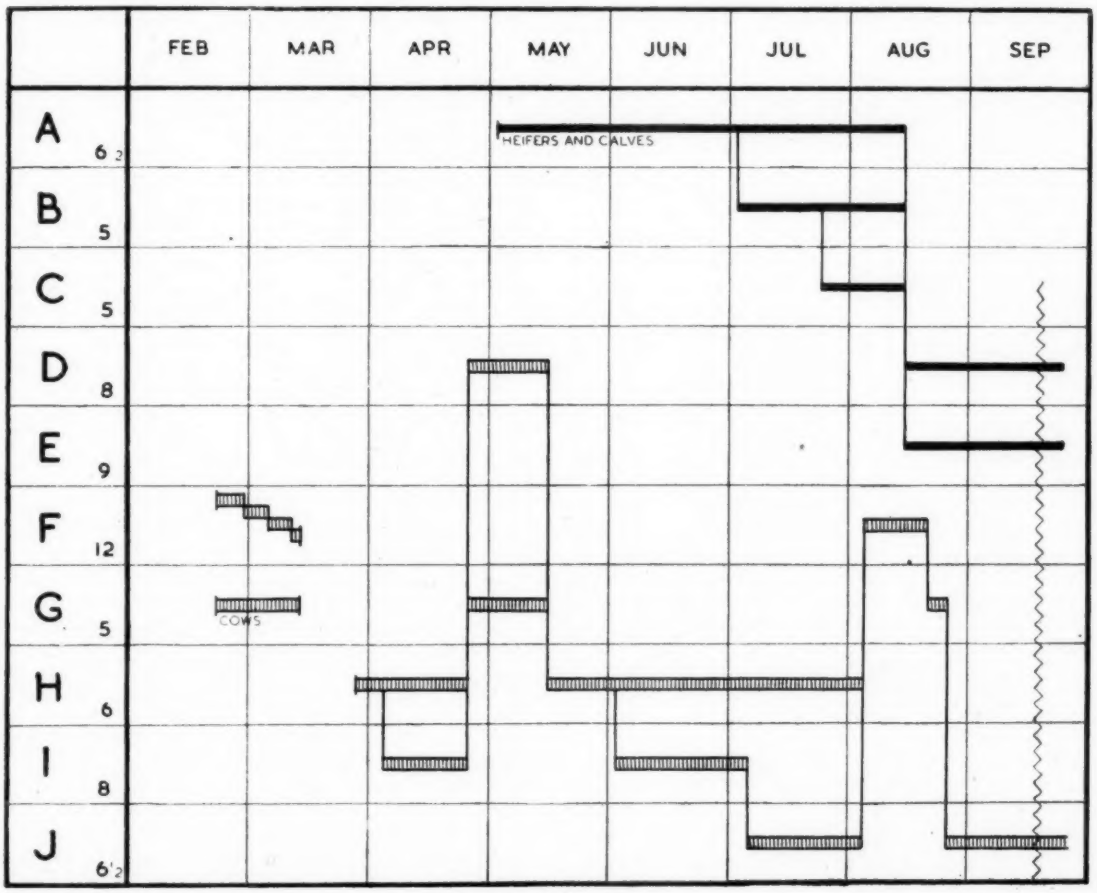


FIG. 15.—Outbreak O.

period of three months between the cows leaving it and the young stock coming in. If they became infected in this way, however, why were the symptoms so long delayed? If the symptoms were brought on by a move to the aftermath E why should the incubation period be so long and why after such a long period did the symptoms appear simultaneously in all five heifers? One thing at least is certain. The appearance of the symptoms in these five animals was associated with their being heavily in-calf. Another interesting point is that the animal that died was evidently capable, at any rate for a time, of carrying an appreciable worm burden without noticeable symptoms.

Outbreak P

Herd of Guernseys, closed many years, had been moved three years before to a new farm. Access to distant fields difficult. Young calves from age of one month ran during day in a small paddock. At other times this accommodated odd animals. At age of six to seven months calves put together with remaining young stock. In May such a group of calves was put with other young stock on a distant ley very rich in S.100 white clover. Remained there three weeks and although grass still growing away from them moved to piece of downland. Thereafter moved from enclosure to enclosure, never staying long in any one. In August returned to ley which had not been grazed since and where there was now an embarrassing amount of keep. Ten days to a fortnight later coughing of an alarming nature began. If anything more severe in older animals. Animals moved but coughing continued. Three months after onset of outbreak two in-calf heifers worst affected, and housed ever since, suddenly developed severe fog fever-like symptoms. Both aborted. A few of the other heifers that calved normally were drafted into the milking herd and three weeks after the arrival of the first a number of cows, principally the younger ones, began to cough mildly.

Outbreak P is of interest in that the two in-calf heifers that had been the most seriously affected, simultaneously developed fog fever-like symptoms though they had been housed for 10 weeks or more. Again, as in outbreak O, an immediate cause of this simultaneous and sudden onset of symptoms is apparently lacking.

As apart from this it is interesting that a ley that was grazed and then left is prominent in the story. It would seem that the management of the half-acre calf paddock was calculated to get every calf lightly infected. These lightly infected calves would naturally be passing larvae and it may be that on the ley to which the young stock were moved, and to which they subsequently returned, conditions for the larvae were much better than on the natural downland. The appearance of symptoms 10 days to a fortnight after the animals returned also fits in very well with this concept.

Outbreak Q

Herd of Guernseys on same farm and managed in same way over 20 years. No female stock introduced 18 years. No clinical husk for five years when an outbreak in yearling heifers. Calves housed in their first year. Dry cows ran with in-calf heifers. A week before ralving they were brought to a group of buildings and can in adjacent paddock (F in Fig. 16). Young stock were yarded in winter but cows ran out winter and summer.

A and B were in continual use by the cows, A by night and B by day, as they had been for 20 years. This system of management had tended to punish the early growing grasses. C was a two-year-old timothy cocksfoot S.100 ley which the stock did not find very palatable. However, it was very prolific and was tall and dense till end of June and still provided a substantial bite end of July. D was a rye-grass S.100 ley in its first year and cut for silage in May. "Cows" numbered 40 in-milk. "In-calf heifers and dry cows" numbered 20, and "bulling heifers" 15. Movement of dry cows to and from in-calf heifers had proceeded at a steady rate but precise details unobtainable.

July 12th, intermittent cough noticed among cows. Within a week one animal acutely dyspnoeic and remainder all coughing violently. Off feed, losing condition and marked fall in milk yields. Four days later more animals with dyspnoea and temperatures between 103° and 104°. July 24th, dry cows grazing with in-calf heifers noticed to be coughing. Continued to do so but in-calf heifers did not cough until after they moved to C in August and then only mildly. Bulling heifers did not cough at all.

The histories of a number of individual cows are indicated in the diagram. 1, Harebell left milking herd June 15th to join in-calf heifers. Noticed coughing July 24th. Died September 8th after being acutely ill less than a week. Lungs grossly oedematous and emphysematous. 2, Ilex had been dry many months and with in-calvers all spring and summer. Noticed coughing July 24th but only mildly. 3, Lil had been in F all spring and summer. Began to cough mildly July 20th. 4, Sally had been housed 60 days when turned out with in-calvers June 23rd. Died August 6th after short fog fever-like illness. 5, Bonny, a first-calf heifer, had been conspicuous as bad doer when a calf. Began to cough end June, fortnight before others. Only animal that did not lose condition and her cough said to be qualitatively different. 6, Dewdrop brought to laboratory and observed. Alternated between periods of dyspnoea and normal respiration with occasional cough. Weak, and finally died September 22nd. Lungs contained 2,300 mature worms and a few immature. Of two faeces samples examined July 26th one negative and other from first cow affected had 952 larvae per 10 grammes. Samples taken September 7th gave following results: Dewdrop 13,400, Harebell 1,215 larvae per 10 grammes. Ilex, Lil and Bonny negative.

Outbreak Q is presented as an example of those outbreaks in which the facts do not yield readily to any interpretation. Although the onset of symptoms was fairly sudden there was no obvious event which might be associated with this. The supposition that lungworm infection was always present in the herd is supported by the outbreak which had occurred in yearlings five years before. The in-calf heifers would therefore be resistant while the bulling heifers, out for the first time, would not. The fact that the bulling heifers did not become infected suggests that there was no source of infection in E during May and early June. On the other hand there can be little doubt that the dry cows

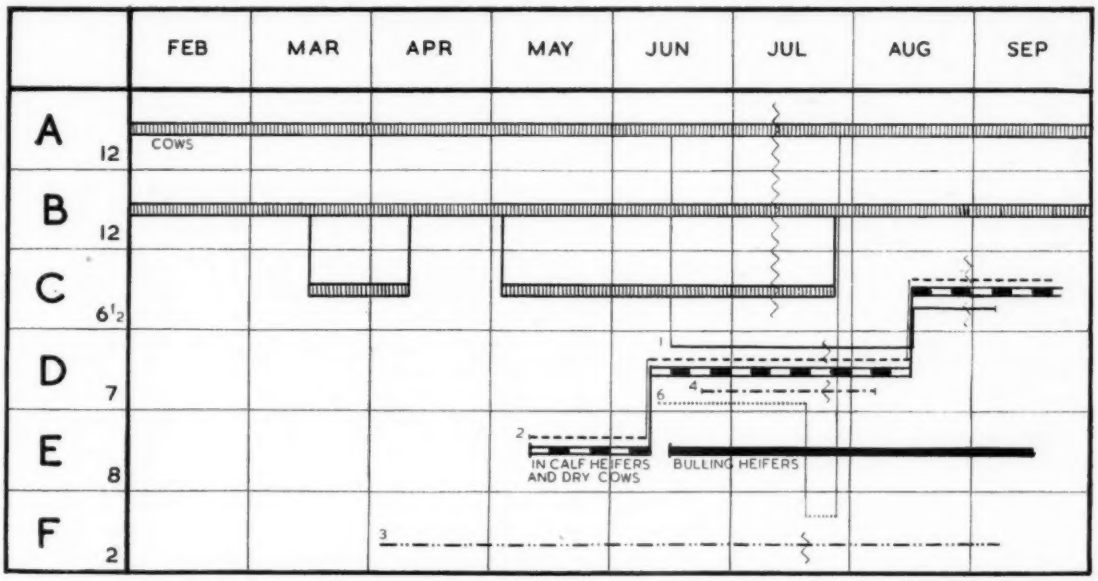


FIG. 16.—Outbreak Q.

became infected while they were in D. The possibility that they picked up the infection before they joined the in-calf heifers may be excluded since Ilex (2 in diagram) had been with the heifers since the winter, while Sally (4 in diagram) had been housed for two months. This latter animal was apparently more susceptible than those which had not been housed. The same phenomenon was noted in outbreak D where the most seriously affected cow was one that had been housed until late in June. Similarly, in outbreak C the most severely affected animals were the heifers which had spent several months in G. These circumstances tend to confirm the view that sudden exposure to infection is the most effective means of establishing an appreciable infection in the animal.

It would appear then that though D was a sufficient source of infection to produce symptoms in the dry cows and to kill one suddenly exposed to it, the in-calf heifers were not affected. Twelve days after being moved to C, however, they did begin to cough though not violently.

Either C was more heavily infected than D, or else it possessed that elusive something which, in aftermaths, seems to precipitate the onset of symptoms. This latter possibility, while it should not be discounted altogether, seems less likely since a number of dry cows which were never in C became very seriously affected without any special stimulus. It would appear to be possible, therefore, that animals which will withstand one level of infestation will show symptoms when transferred to one more intense.

It will be noticed that the cow Lil (3 in Fig. 16), which had been in the paddock F, began to cough about July 20th. As animals calved they were put in this paddock and it would follow from this that some animal disseminating larvae in its faeces brought the infection with it. The latest that this carrier animal can have come into F would seem to be the beginning of July. Now all animals spent some time with the in-calf heifer before they came into F, but it would appear that there was no source of infection among the dry cows and heifers during May. It would follow, therefore, that the infection was brought to F by an animal, presumably a dry cow, that began to pass larvae during June. If there were such animal in the milking herd during June, the level of herbage infestation on the cow grazings cannot have risen until after the middle of that month since Harebell (1 in diagram) evidently did not pick up the infection until after she was put in D.

That the sudden appearance of symptoms was not apparently related to any move need not invalidate the thesis that the cows were exposed to heavy infection fairly suddenly. According to the herdsman the milking cows spent some time in C each day during May and June but found it unpalatable and grazed very little. It was only towards the end of June that they were driven by the shortage of keep on A and B to graze C hard.

We would suggest, therefore, that during May and most of June the cows were infecting C though not grazing it and that the lush grass provided very suitable conditions for the larvae.

Conclusions

No single simple feature appears to be common to all outbreaks and no simple explanation or train of causation is applicable to them all. Nonetheless, it is felt that some trace of a pattern is discernible and a number of indications emerge.

Perhaps the most striking point is the frequency with which symptoms appear approximately 12 days after the animals return to a field offering an excess of keep. In an experimental study of the fog fever-like syndrome seen in parasitic bronchitis (Michel, 1954), it was found

that one of the stages in a lungworm infection in which this syndrome appears is 10 to 13 days after the administration of larvae to animals that had had previous experience of infection. In many outbreaks that have been discussed this interpretation would appear to fit. In other outbreaks, however, of which K and L are examples, extreme symptoms appeared 12 days or less after a move to aftermaths which were certainly clean, and it was concluded that these aftermaths had in some way precipitated the onset of symptoms.

The second point that emerges is that in the great majority of outbreaks the infection was endemic on the farm, generally in a subclinical form ; the third that though adults certainly acquire a resistance to the parasite, as do younger cattle also, they may, nevertheless, become infected, presumably if they are exposed to a sufficiently great uptake of larvae. Whether any external factors may operate to render them susceptible does not emerge from this investigation, but inadequate nutrition would not appear to be such a predisposing factor. Even adult cattle can become infected if the dose of larvae is great enough, but should they have been withheld from infection altogether for some time then they will succumb to a small dose. The longer they are not exposed to infection the more susceptible they will be. Again, the severity of the infection that animals will acquire is dependent within certain limits on the level of infection to which they are first exposed ; not only must infection be heavy to succeed in becoming established in the adult but it must also be relatively sudden.

The ultimate source of infection is in the small numbers of larvae disseminated in the faeces of carrier animals which may themselves show no symptoms. One of the conclusions from a study of the inhibition of development of lungworms, an account of which is in the hands of the publishers, was that any animal that has at any time been exposed to lungworm infection represents a potential source of infection. Furthermore, a consideration of the outbreaks tends to confirm the view that the larvae do not survive on the ground over winter as they provide numerous examples of animals remaining free from infection while on fields that have carried no cattle for four months or longer, but becoming infected when moved to fields that have been occupied by other bovines more recently than this. Experimental evidence in support of this view is provided by Wetzel (1950) and also by Michel & Parfitt (1955).

From the evidence here presented it would appear that the small numbers of larvae disseminated by carriers may be turned into a dangerous level of infestation on the ground in two ways. The infection may be augmented or built up by the action of susceptible animals, usually calves. These, when exposed to a low level of infestation, become relatively lightly infected, yet pass sufficient larvae in the faeces appreciably to raise the level of infestation of the pasture. Yet the resistance that they acquired through carrying that light infection appears to protect them against the higher level of herbage infestation which they have been instrumental in creating.

In many cases, however, the carriers appear to give rise to such a dangerous infestation directly without the intervention of susceptible animals as a " culture medium." The conditions which appear to make the small numbers of larvae passed on to the ground most effective would seem to exist in lush or dense long herbage such as a vigorously growing ley or a sward rich in clover. These indications have been confirmed and amplified by laboratory observations (Michel & Rose, 1954), from which it would appear that diarrhoeic faeces scattered in lush herbage represent a particularly dangerous situation.

There can be little doubt that control of parasitic bronchitis should be by husbandry practices but when the great diversity of the circumstances is considered in which husk outbreaks in adults occur, the difficulty of making realistic recommendations appears formidable.

The aim of husbandry would seem to be to keep the cattle constantly exposed to a low level of infection and to avoid sudden exposure to massive infections. If, as we have seen, such massive infection may be brought about by the action of susceptible calves or by carriers on lush ley pastures, then both these factors should, as far as possible, be avoided. To run the calves separately from the milking herd could be easily arranged on almost any farm, but to avoid rapidly growing herbage would run counter to the objects of grassland management. To suggest that the grass should grow to the stock rather than away from it may appear a counsel of perfection, but such is and should be the aim of the conscientious grazier. At least it should be possible to avoid putting the stock into tall grass if it has been grazed less than four months before.

In so far as a number of outbreaks seem to be associated with grazings strictly reserved for the milking herd, one is tempted to suggest that such an arrangement whereby " the cows march westward and the young stock eastward " may lead to the formation of a worm-free susceptible community on the milking side and a subclinically infected one on the young stock side. It would follow from this that cow pasture represents a possible danger. To graze heifers, but not calves together with the cows, might well be regarded as an unrealistic suggestion since the milking cows need the best pastures, and young stock grazing on these would tend to acquire an undesirable configuration. The practice of allowing older heifers to clear up behind the cows may therefore have some advantages though it appears to have had unfortunate consequences in outbreak R.

The conclusion seems to be inescapable that as yet no recommendations can be formulated with confidence.

THE CLINICAL MANIFESTATIONS OF HUSK IN ADULT CATTLE

Taylor (1953) distinguished between typical husk as seen in the calf and characterised by coughing, and atypical husk as seen in adults and characterised by pulmonary oedema and emphysema. This classification would appear to be rather too rigid. Two syndromes do certainly occur but a single individual may exhibit both at different times during the course of the disease, even alternately in rapid succession, and different individuals in the same herd or group may show either syndrome. Moreover, all the manifestations of the disease in the cow may also be seen in the calf and yearling.

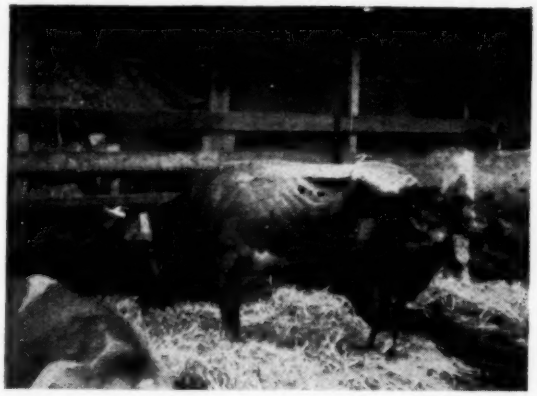


FIG. 17.—Acute parasitic bronchitis in a Guernsey heifer.

The old concept that "animals with husk must cough" can no longer be accepted. Coughing is a symptom of mild husk, while the outstanding feature of acute husk is dyspnoea. In such cases the cough may be entirely absent. This is as true of the calf as it is of the adult. The acutely dyspnoeic animal is unable to cough; the less severely affected animal may cough and pant intermittently while the mildly affected animal will cough without panting.

The great range of variation both in the histories of individuals and in the herd picture is exemplified in the outbreaks presented on preceding pages. In some individuals there is coughing before the onset of dyspnoea but in others this appears to be absent. Dyspnoea may begin either late or early during the course of the disease.

In the herd or group those animals which subsequently turn out to be the most severely affected are frequently the first to show any symptoms. They may either cough or their sudden acute illness or even death may be the first sign of an impending outbreak. The less severely affected animals will often develop symptoms later and will then only cough. One may think in terms of a continuous range of symptoms of increasing severity from an occasional mild cough to acute respiratory distress with pulmonary oedema and emphysema. This extreme syndrome closely resembles the condition known as fog fever and described by Barker (1948), Begg & Whiteford (1948) and by Leslie (1949). This fog fever syndrome when associated with parasitic bronchitis is seen most frequently in adults but may be encountered in young stock also. Thus, in outbreak B, it was seen in yearlings, while in outbreak A the chief symptom in the calves was acute dyspnoea. In some outbreaks the severe symptoms of the fog fever syndrome were preceded by a few days during which there was some loss of condition and a slight drop in milk yield, but as a rule the syndrome was characterised by the sudden onset of acute dyspnoea. Once this appears the animal presents a picture of extreme distress, and conveys the impression that death from oxygen starvation might supervene at any instant.

A large proportion of affected animals have a loud expiratory grunt and breathing is rapid, shallow and abdominal. Each rib is held out rigidly so that it shows clearly under the skin and the costal arch is especially prominent. In characteristic posture the head and neck are stretched out low with the mouth open and the tongue protruding. Feeding and drinking stop immediately the severe symptoms develop but the impression is not so much that appetite and thirst have been lost but that oxygen is the urgent need and there is no time for eating or drinking or even coughing. As every effort must be concentrated on forced breathing the animal strongly resents being moved.

In the lactating animal the milk yield is greatly depressed and the temperature fluctuates but may be as high as 106° F. Cows and heifers in the later stages of pregnancy may abort. Some animals die after a very short spell of severe dyspnoea while others live on for days and weeks in an inexplicable manner. Such animals become very dehydrated and hidebound and at *post-mortem* examination their rumens contain a dried mass of friable fibre. It is doubtful if all such animals would die if some form of forced feeding could be adopted.

The speed with which affected animals lose weight and condition is spectacular. Closer examination of the animals often shows that the upper half of the thorax is abnormally resonant, while the lower half is dull and the *post-mortem* distribution of lesions corresponds with this. On auscultation highly characteristic sounds may be heard, associated with interference with lung function due to the presence of gas and oedema fluid in the interstitial tissue. These sounds may vary from a continual crackling crepitation to a "musical box" or a mixture of both while incessant grunting may obscure all.

At *post-mortem* examination the first striking feature is that the lungs are blown out and have not collapsed. The lung substance is found to be surprisingly heavy and there is a marked degree of oedema and emphysema in the interlobular septae, the oedema being particularly marked around the lower part of all lobes. Small and large bubbles of gas are everywhere throughout the interstitial tissue. In cases of longer standing areas of consolidation with alveoli distended by a yellow exudate may be present.

Recovery may be complete in as little as three weeks but more severely affected animals may remain desperately ill for months. It is, however, impossible to predict whether an affected animal will live or die and it is, therefore, unwise to send any animal to the knackery unless it is dead or unmistakably *in extremis*. It is our experience that many animals are slaughtered which, with patience and careful nursing, might have recovered.

Summary and Conclusions

1. Seventeen outbreaks of husk in adults and their histories, selected to exemplify the greatest diversity, are described and discussed. A number of indications emerge.

2. The nature of the symptoms of husk varies according to their severity. At one extreme a mild cough is encountered, at the other a syndrome identical with that seen in fog fever. This syndrome is described.

3. The onset of symptoms is generally sudden and almost simultaneous in all the animals affected.

4. Symptoms occur :—

(*a*) Twelve days or more after exposure to heavy infection.

(*b*) Twelve days or less after a move to aftermath.

(*c*) Without any obvious precipitating cause.

5. The various outbreaks have no simple factor in common.

6. There is no evidence of any factor rendering adult animals particularly susceptible. The plane of nutrition does not appear to play any part.

7. Sudden exposure to infection is more dangerous than gradual exposure. An animal that has been entirely withheld from infection for some time appears to be particularly susceptible.

8. In most of the outbreaks the infection was endemic on the farm, but at a subclinical level.

9. There is considerable circumstantial evidence that the ultimate source of infection is in the form of larvae passed by carrier animals and not larvae that have persisted on the ground through the winter. The indications are that the larvae do not persist on the ground even as long as five months.

10. The small numbers of larvae disseminated by carrier animals may give rise to a dangerous level of infestation on the ground in two ways :—

(*a*) By the intervention of calves who become lightly infected but pass a greatly increased number of larvae in their faeces.

(*b*) Directly where the conditions are particularly favourable for the larvae.

11. Lush leafy leys appear to represent a particularly favourable vehicle for the survival and transmission of infection.

12. The level of infestation on the ground may remain dangerous even though an intervening hay cut has been taken.

References

BARKER, J. R. (1948). "Conference on Grassland and Animal Health." N.V.M.A. Publication 17.

BEGG, H., & WHITEFORD, W. A. (1948). Acute Interstitial Pulmonary Emphysema of Bovines. *Vet. Rec.* **60.** 135.

LESLIE, V. J. S. (1949). Fog Fever. *Ibid.* **61.** 228.

MICHEL, J. F. (1954). A Contribution to the Aetiology of Fog Fever. *Ibid.* **66.** 381.

———, & PARFITT, J. W. (1955). A Contribution to the Epidemiology of Parasitic Bronchitis in Calves. *Ibid.* **67.** 229.

———, & ROSE, J. H. (1954). Some Observations on the Free Living Stages of the Cattle Lungworm in Relation to their Natural Environment. *J. Comp. Path. & Therap.* **64.** 195.

TAYLOR, E. L. (1953). Parasitic Bronchitis in Cattle. *Vet. Rec.* **63.** 859.

———, & MICHEL, J. F. (1952). Inhibited Development of Dictyocaulus Larvae in the Lungs of Cattle and Sheep. *Nature.* **169.** 753.

WETZEL, R. (1950). *Zur Epidemiologie des Lungenwurmbefalls bei Rindern Monatshefte für Veterinärmedizin.* **3.** 141.

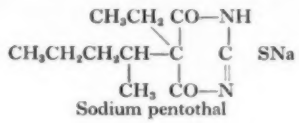
Surital Sodium: A General Anaesthetic for Use in Small Animal Surgery

BY

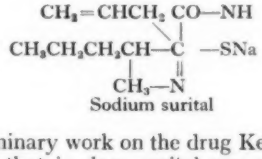
H. A. CRAWSHAW

Newbury, Berks

FOR some time a new thiobarbiturate, surital sodium (5-Allyl-5-(1-methylbutyl)-2-thiobarbituric acid) has been in use in America, and recently a supply was obtained for trial.* It is similar in composition to pentothal sodium except that an allyl group is substituted for the ethyl side chain.



Sodium pentothal



Sodium surital

In preliminary work on the drug Kelly *et al.* (1948) considered that in dogs surital was more potent but less cumulative than pentothal. Using pentothal as a standard, they found surital to be 139 per cent. as potent. Later work by Wyngaarden *et al.* (1949) indicated that surital had a potency of 1.5 compared with 1.0 for pentothal, based on the length of anaesthesia from the onset of unconsciousness until the dog was able to stand erect after the intravenous administration of the drugs. These workers found that surital produced a more rapid induction of anaesthesia than pentothal with fewer signs of excitement and a more rapid recovery. Being more potent, a smaller total dose was required with a consequent shortening of the time for detoxication. Unlike some workers in the human field, who considered that in their patients laryngo-spasms was less with surital than with pentothal, Wyngaarden *et al.* found no significant reduction of laryngeal reflex activity in cats with the former drug.

Materials and Method

Surital is an almost white flocculent crystalline substance readily soluble in water, having a strong sulphurous or garlic-like odour. Solutions have been used a week after being prepared, without any noticeable variation in potency, although after this time a slight precipitate occurs. This is said to be due to the absorption of CO_2 from the air, which lowers the *p*H of the solution (Eads, 1953). The injection of air into the bottle from the syringe therefore should be avoided, and neither should glucose, buffer agents, etc., be used in the solvent as these may hasten precipitation.

The makers recommend that 1 gramme should be dissolved in 40 c.c. of water to make a 2.5 per cent. solution and it seems that many workers use this

* From Messrs. Parke, Davis & Co.

dilution while others employ 4 per cent. solutions. In the writer's hands the use of a more concentrated solution has been entirely satisfactory, and allows the use, except in very large dogs, of a 5 or 10 c.c. syringe. One gramme of sodium surital is dissolved in 15 c.c. of water, giving an approximate concentration of 1 grain in a cubic centimetre.

Clinical Observations

It has been found that the anaesthetic dose, as judged by the loss of the pedal reflex, has varied considerably in individual animals. On the average, the slower the injection is given the greater the dose which can be expected to be used and the longer the duration of anaesthesia. This fact, of course, applies to pentothal sodium and the other barbiturates. In the original American work the dose was injected at constant speed of 3 c.c. of a 2.5 per cent. solution per minute (Reutner & Grumzit, 1948), but in practice, in surgical cases this is not always practicable, as allowance has to be made for the condition and anaesthetic acceptance of individual animals.

In the present series of observations approximately one-third of the estimated dose was given fairly rapidly. This eliminates any fear which the animal might show upon losing consciousness and ensures a smooth induction of anaesthesia. The recovery of consciousness was almost invariably quiet and the paddling with the feet which sometimes accompanies recovery from other forms of anaesthesia was seldom noted.

A total of 37 dogs and eight cats have been anaesthetised with surital sodium, some for the purpose of euthanasia but the majority for surgical interference. The ages of the dogs varied from seven months to 16 years, and the weight from 14 lb. to 70 lb. The average dose was found to be 1/6th of a grain per lb. (11 mg. per lb.) for dogs and between 1/6th and 1/7th gr. per lb. (10 mg. per lb.) for cats. No deaths in surgical cases have been attributable to the anaesthetic. Borgman (1953) found that 8.3 mg. per lb. gave an adequate depth of anaesthesia, while nine dogs which were given a toxic dose, averaging 17.5 mg. per lb. survived without stimulation. Reutner in his series of observations found that 1/8th gr. per lb. (8.1 mg. per lb.) gave good anaesthesia.

Discussion

It is considered that variation in the speed of injections may account for the difference between the present observation and those of the Americans. Their standard rate of injection does not seem to take into account individual idiosyncrasies. A slow injection rate explains the apparent anomaly of older dogs occasionally taking a higher dose of the drug. In these cases, as a precaution, the anaesthetic was administered very slowly, and detoxication proceeded as it was being injected.

In the writer's hands the induction time, using surital sodium, averaged five minutes and did not differ significantly from that of pentothal sodium. Using a medium depth of anaesthesia there was generally some slight return of reflexes in 10 minutes. When the initial anaesthesia was deeper, 15 to 20 minutes elapsed before any significant return of reflexes was noted. When it was necessary to prolong anaesthesia, ether was employed, and in these cases comparatively little was necessary to maintain good anaesthesia.

The recovery stage varied considerably and depended to some extent upon the duration of the operation, additional anaesthetic administered and the degree, if any, of surgical shock. When surital was used alone as a single injection for short operations the patients were usually on their feet within 30 to 40 minutes from the completion of the injection.

The present observations confirm the American work that the induction of anaesthesia in both dogs and cats is quiet, as is the recovery stage. The impression is that the rather violent spasm seen in many cats when handled during the latter stage of pentothal sodium is less frequent when using surital sodium, although insufficient cases have as yet been observed definitely to establish this point. Given slowly the anaesthetic is less depressant to the respiratory centre than pentothal sodium and fewer cases of apnoea have occurred.

Surital was first used here for euthanasia and it was found that even when a dose considerably larger than that necessary for anaesthesia was injected the heart continued to beat strongly, and frequently breathing recommenced after three or four minutes without artificial respiration. With this experience no alarm has been felt in the few cases where breathing has been in temporary abeyance during the induction phase when used in surgical cases. In each instance the heart has continued to beat strongly and a short period of artificial respiration has been sufficient for resuscitation. In these cases it is probable that the injection had been given too quickly. Ether has been used to continue the anaesthesia when the initial injection of surital has not been sufficient for the whole duration of the operation. It is the practice to premedicate the patients with an appropriate subcutaneous dose of atropine sulphate, as it was found that in some cases with surital alone, salivation was profuse.

Summary

1. Surital sodium (sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbituric acid), a short acting barbiturate anaesthetic has been studied in 37 dogs and eight cats.

2. An average dose of 1/6th gr. per lb. (mgm. per lb.) administered intravenously has been found to give good surgical anaesthesia in dogs, and between 1/6th and 1/7th gr. per lb. in cats.

3. The induction and recovery stages are quiet. It is considered that surital sodium is slightly less depressant to the respiratory centre than is pentothal sodium.

4. No deaths from respiratory failure occurred in 31 surgical cases.

5. To control excessive salivation premedication with atropine sulphate is recommended.

Note: Surital sodium is not at present available in this country.

(Concluded at foot of column 1 overleaf)

News and Comment

CONTAGIOUS ABORTION: SUPPLY OF MINISTRY S.19 VACCINE

It has not been practicable hitherto to supply the Ministry's S.19 anti-abortion vaccine from Animal Health Divisional Offices except to Local Veterinary Inspectors for use under the official Calf Vaccination Scheme. Veterinary surgeons requiring vaccine for their private practice have been required to obtain it by post from the Ministry's Veterinary Laboratory at Weybridge, Surrey.

From April 1st, 1955, the Ministry will be able to supply this vaccine from its Divisional Offices. All veterinary surgeons should therefore from April 1st order their requirements of Ministry S.19 vaccine (either by letter or telephone) from the local Divisional Veterinary Officer and not from Weybridge. Alternatively, if more convenient to the veterinary surgeon, he may call and collect it at the Divisional Office.

A supply note indicating the quantity and date of issue will be included with each batch of vaccine supplied by the Divisional Office on and after April 1st, 1955, and the cost of vaccine so supplied will be included in the quarterly account rendered by the Veterinary Laboratory, Weybridge, for goods supplied direct by the Laboratory.

As the Ministry is unable to allow credit for any returned unused vaccine only sufficient vaccine for forseeable needs should be ordered. The vaccine has a "life" of four weeks from the date of supply by the Divisional Office; the expiry date is stamped in red ink on each vaccine bottle.

R.C.V.S. LIBRARY

The R.C.V.S. Library will be closing for the Easter vacation from 2 p.m. on Thursday, April 7th until 10 a.m., the usual opening time, on Tuesday, April 12th.

References

BORGMAN, R. F. (1953). Dosage Studies of Surital Sodium in Dogs. *J. Amer. Vet. Med. Assoc.* **122.** 161.

EADS, F. E. (1953). Surital Sodium Anaesthesia in Dogs and Cats ; A Symposium Covering 7,522 Patients. *Calif. Vet.* **7.** 1.

HELRICH, M., PAPPER, E. M., & ROVERSTINE, E. A. (1950). Surital Sodium : A New Anaesthetic Agent for Intravenous Use. Preliminary Clinical Evaluation. Anaesthesiology. **11.** 1.

KELLY, A. R., SHIDEMAN, F. E., & ADAMS, B. J. (1948). Comparison of Blood Levels of Thiopental (Pentothal), 5-allyl-5-(1-methylbutyl)-2-thiobarbituric Acid (Surital) and Thioethamyl in the Dog. *Federation Proceedings.* **7.** 233. No. 1.

PHILLIPS, H. S. (1953). Some Clinical Observations on the Use of Surital Sodium in Combination Anaesthesia. *Current Researches in Anaesthesia and Analgesia.* **32.** 1.

REUTNER, T. F., & GRUMZIT, O. M. (1948). Surital Sodium, A New Anesthetic and Hypnotic. *J. Amer. Vet. Med. Assoc.* **113.** 357.

WYNGAARDEN, J. B., WOODS, L. A., REDLEY, R., & SEIVERS, M. H. (1949). Anaesthetic Properties of Sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate (Surital) and Certain Other Thiobarbiturates in Dogs. *J. Pharmacol. Exper. Therap.* **95.** 322.

IN PARLIAMENT

Veterinary Medicines and Drugs

Sir A. Bossom (March 24th) asked the Chancellor of the Exchequer how far Purchase Tax is charged on dispensed veterinary medicines and drugs as it is on proprietary brands; and if he will make a statement.

Mr. H. Brooke : Medicines and drugs which are of established therapeutic value can be obtained for veterinary dispensing free of tax.

Sir A. Bossom asked the Chancellor of the Exchequer if, in his forthcoming Budget, he will consider removing the Purchase Tax from veterinary medicines in order to permit farmers to take full advantage of measures for the prevention of disease and to cure illness in their stock.

Mr. H. Brooke : I cannot anticipate my right hon. Friend's Budget statement. At present animal medicines are dealt with broadly on the same lines as human medicines, with the result that a large number are exempt.

AWARD TO VETERINARY SCIENTIST

The Poultry Association of Great Britain initiated three years ago the presentation each year of not more than three awards for distinguished service in, or to, the poultry industry over a long period of years. The award consists of a bronze medal and parchment citing the award. A distinguished recipient of one of these awards this year is Mr. J. E. Wilson, B.SC., M.R.C.V.S., F.R.S.E.

HOLIDAY EXCHANGE

There appear to be a number of French veterinary surgeons whose sons and daughters would like to spend part of their summer holidays with British veterinary surgeons and, in return, would like to entertain the British children in their homes.

The following is a list of places where there is a French veterinary family who would like to make this exchange, and any member of the profession who is interested is asked to write as soon as possible to the General Secretary of the B.V.A. for further particulars.

Loiret (Châteaux de la Loire Region): Boy of 13.

Drôme Region : Boy of 15 would like to visit England about July 10th and take back an English boy in August.

Drôme Region : Two girls, free on August 5th, would like to stay *au pair* with one family, or two families near to one another. The elder girl, who is 19, would teach children and help in the house; the younger, 18, would look after children and help in the house.

Yonne Region : Boy of 15.

Gers Region : Boy of 13.

Gers Region : Boy of 13.

Sarthe Region: Boy of 17 would like either to visit *au pair* or receive an English boy in exchange.

Vienne Region: Boy of 14. His mother would bring him and would return to France with an English boy.

Landes Region: A young man of 20, who has just finished a year specialising in mathematics. He would like to stay as a paying guest, but would take another boy to his home in exchange if desired. He would like to come to England from about July 15th to August 15th.

Seine et Oise Region: Boy of 17 living in Versailles.

Paris: Girl aged 14.

Spanish Veterinary Student: A 26-year-old veterinary (final year) student, Pedro Cangas, of Plaza Independencia No. 2, Madrid, wishes to work on a British farm for the summer months, offering his services in order to improve his English. Any reader who may be good enough to help or advise this young man is asked to write to him direct.

SHEEP SPRAYING

Since Tessier elaborated the first successful sheep dip in 1810, it has been the practice all over the world to dip sheep in a bath for the control of external parasites. The types of bath have varied from a small hand-tub for the treatment of individual sheep to long-swim baths for the dipping of several thousand animals.

This method of treatment has been so successful that it has been carried out for many years without any material alteration taking place in the installations and methods of immersion. However, the introduction of the newer insecticides, such as DDT and BHC, introduced new standards of parasite control and also revealed several unsatisfactory features about the typical dipping bath.

These insecticides had frequently to be used as suspensions and if employed in poorly designed dipping baths, or improperly calibrated baths, the efficiency of the insecticide was impaired. It was also discovered that when these insecticides were formulated without the presence of a bacteriostat, bacteria present in the bath were able to multiply and, when baths were left standing for several days, the bacterial population was sufficient to cause infected sores and lameness due to *Erysipelothrix rhusiopathiae*.

It became obvious therefore that to obtain the maximum from these new insecticides there would be a great advantage to be obtained by using fresh wash through a spray race. Several forms of spray race were tested but, when using such insecticides as DDT and BHC, the residual effect on the sheep was poor.

In 1954, when Dieldrin became available for the treatment of the parasites of animals, it was found that this insecticide appeared to give very good residual action on the fleece of sheep in the control of maggot-fly, even when put on in very small quantities. These findings stimulated further investigation in the use of a spray race designed primarily for maggot-fly control.

A suitable spray race has now been developed by the Cooper Technical Bureau in association with Messrs. E. Allman & Co. Ltd.

This race was demonstrated at the Cooper Field Research Station at Berkhamsted on March 30th, and a feature of its use is that up to 40 to 60 sheep can be treated per minute and each sheep receives approximately 1/6th gallon of insecticide. The race so far has only been tested against the sheep maggot-fly, and would appear to be very useful indeed for this purpose.

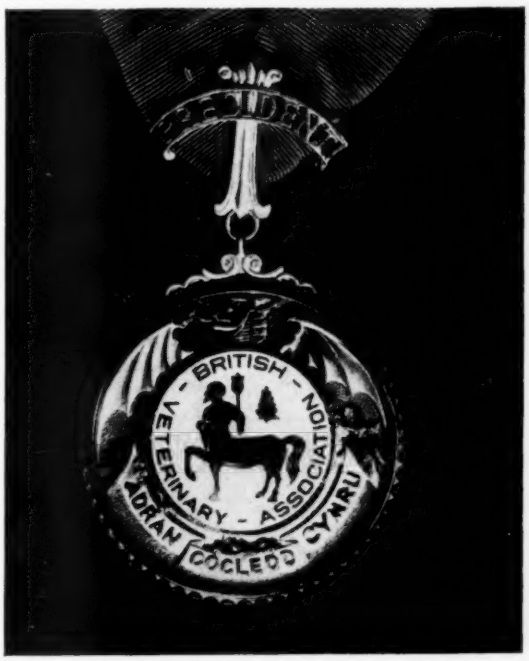
THE NORTH WALES DIVISION

The North Wales Division has adopted the design shown in the photograph below of the medallion for its crest. The B.V.A. graciously gave consent to use the parent body's badge which forms the central portion of the crest, in the form of a silver Centaur with silver lettering on a background of blue enamel. The remainder of the design—a Welsh dragon supporting the central badge with a scroll bearing the words "Adran Gogledd Cymru" (North Wales Division) is in silver. On the reverse side the words "founded 1900" are engraved.

The medallion was presented to the Division by Mr. T. W. Hughes, of Mold, in memory of his father, Mr. W. Hughes, of Caerwys, and an inscription to that effect appears on the case in which the medallion is kept.

At a meeting of the Division held on March 11th, the pendant was presented to the Division.

Suitable expressions of gratitude, to be conveyed to Mr. T. W. Hughes for his kindness and generosity were made by the President and others.



BRIGADIER A. G. RALSTON

Brigadier A. G. Ralston, M.R.C.V.S., assumed the appointment of Director, Army Veterinary and Remount Services with effect from March 24th, 1955. This officer was lately serving in Singapore.

VETERINARY SURGEON LEADS R.AUX.A.F. SQUADRON

When the Secretary of State for Air recently opened 610 Squadron's new town headquarters at the R.A.F. station at Hooton Park, he watched an impressive fly-past of the Squadron's 16 Meteor fighters.

This Squadron, which is attached to the Royal Auxiliary Air Force, is led by Squadron-Leader J. Storrar, D.F.C., a veteran of the Battle of Britain. He is a local veterinary surgeon.

AILMENTS OF BIRDS, RABBITS, GOLDFISH, ETC.

There appears to be an increasing demand for veterinary assistance in dealing with the ailments of less usual domestic pets such as birds, rabbits, goldfish, etc. It would be helpful if readers who are interested, or who have specialised knowledge, would kindly notify the General Secretary, British Veterinary Association, 7, Mansfield Street, Portland Place, London, W.1, and at the same time state whether they would be prepared to give advice on specific problems to veterinary surgeons who may require their assistance.

A list of names will then be kept at B.V.A. headquarters, and those wishing for advice are invited to apply to the Association.

PERSONAL

Births

FELL.—On March 21st, 1955, to Margaret (*née* Jackson), wife of B. F. Fell, M.R.C.V.S., at Dundee Royal Infirmary, a daughter, Susan Margaret.

SALISBURY.—On February 13th, 1955, at Bothomes Hall, Whaley Bridge, Stockport, to Isobel (*née* Murray), wife of Norman Salisbury, B.SC., M.R.C.V.S., a son, Neil.

TEAKLE.—On February 7th, 1955, at 20, Ferrers Road, Lewes, Sussex, to Mary (*née* Burns), wife of John Teakle, M.R.C.V.S., a daughter, Katherine Mary Jane.

MR. H. M. STUCHBERY

Mr. W. G. Pevie, Assistant Director of Veterinary Services, Tanganyika Territory, writes:—

Mr. H. M. Stuchbery, a former member of the Colonial Veterinary Service, who had been farming in the Northern Province met a tragic death at the hands of local tribesmen recently.

The death of "Stuch," as he was affectionately called, has profoundly shocked all who knew him. Harold Morton Stuchbery was a typical Australian, full of the joys of life but possessed of a profound sense of duty. He served in the Colonial Veterinary Service for 26 years, being stationed in Fiji, Tanganyika and Nyasaland. It was to Tanganyika he returned when he retired from the Service in 1952 to take up a piece of land in the Northern Province.

Although no longer a young man he had the spirit of youth. He applied himself diligently to the developing of his none too friendly acres with the aid of a small dairy herd and a few good beef stock. It was in the evening whilst wandering around on his small domain that he came on some local cattle trespassing on his land. He challenged the herdsmen and whilst disarming them was struck down with a spear.

It was a tragic end for a man who had by practice and precept done so much for the African and his stock. To his wife and daughter we extend our sympathy and understanding in this their dark hour.

COMING EVENTS

April

2nd (Sat.). Annual Dinner of the B.R.X. Club, at the Royal Veterinary College, 7 p.m.

7th (Thurs.). Spring Meeting of the North of Scotland Division, B.V.A., at the Northern Hotel, Aberdeen, 2.30 p.m.

14th (Thurs.). Annual All-day Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 11 a.m.

15th (Fri.). General Meeting of the Midland Counties Division, B.V.A., at the Star Hotel, Worcester, 2.30 p.m.

20th (Wed.). Meeting of the Royal Society of Medicine, Section of Comparative Medicine, at the Society's house, 5 p.m.

21st (Thurs.). General Meeting of the Herts & Beds Division, B.V.A., at 70, Holywell Hill, St. Albans, 7.30 p.m.

23rd & 24th (Sat. & Sun.). Annual Reunion Weekend for 1952 R.V.C. graduates and contemporaries. Dinner at "Ye Miller of Mansfield" Hotel, Goring, Saturday, 7.30 p.m.

30th (Sat.). Annual General Meeting of the Association of State Veterinary Officers in the Auditorium of the Wellcome Research Institute, 183-193, Euston Road, N.W.1, 1.30 pm. Annual Dinner at the Criterion Restaurant, Piccadilly, W.1, 7 p.m.

May

3rd (Tues.). Afternoon Meeting of the Eastern Counties Division, B.V.A., at Colchester.

6th (Fri.). Royal Veterinary College 163rd Annual Ball at the Hyde Park Hotel.

11th (Wed.). Evening Meeting of the Eastern Counties Division, B.V.A., at Ipswich.

Meeting of the Royal Veterinary College Medical Association at Streatley House, Berks, 2 p.m.

12th (Thurs.). R.C.V.S. Dinner, Dorchester Hotel. Further details will be published next week.

14th (Sat.). Annual Sports Day of the Royal Veterinary College to be held at Streatley, 2 p.m.

31st (Fri.). Annual Dinner-Dance of the Chiron Club in the Royal Hotel, Bristol, 7 p.m.

June

10th (Fri.). Annual Dinner of the R.A.V.C. Officers' Club, at Grosvenor House, Park Lane, London.

September

4th to 10th. 73rd Annual General Meeting and Congress of the British Veterinary Association at Belfast.

APRIL MEETINGS OF COUNCIL AND COMMITTEES, B.V.A., IN EDINBURGH

AT THE ROYAL (DICK) SCHOOL OF VETERINARY STUDIES

Monday, April 4th.—

10.15 a.m. Veterinary State Medicine Committee.
2 p.m. Home Appointments Committee.
4 p.m. Organising Committee.

Tuesday, April 5th.—

10.15 a.m. Parliamentary & Public Relations Committee.
11.30 a.m. Finance Sub-committee.
2 p.m. General Purposes & Finance Committee.

Wednesday, April 6th.—

10.30 a.m. COUNCIL MEETING.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ayr. Kerslochmuir Farm, Dalry (Mar. 21).

Berks. Corporation Farm, Abingdon (Mar. 24).

Cumberland. Row Head, Sowerby Road, Carlisle (Mar. 21).

Dorset. New Barn Farm, Woodlands, Wimborne (Mar. 22); Southwood Farm, Arrowsmith Road, Canford Magna, Wimborne (Mar. 24).

Durham. Welbury Farm, Newton Aycliffe, Darlington (Mar. 21).

Norfolk. Sankence Lodge Farm, Aylsham, Norwich (Mar. 21); Whitehouse Farm, Kettlestone, Fakenham (Mar. 23).

Notts. Brookside, Bawtry Road, Blyth, Worksop Mar. 18); Stud Farm, Weston, Newark (Mar. 19); Laurels Farm, Stanton on the Wolds (Mar. 21); Saucethorpe Farm, Kinoulton (Mar. 22); Holly Farm, Rolleston, Newark (Mar. 24).

Yorks. Carr House Farm, Cayton, Scarborough (Mar. 18); South Farm, Blacktoft, Goole; Wayworth Farm, Commondale; Smallholding, 9, Allendale Road, Sprotsborough Road, Doncaster (Mar. 19); 47, Littleworth Lane, Monk Bretton, Barnsley (Mar. 23); Poplar Farm, Langthorne, Bedale; Smallholding, Redhill, Spofforth, Harrogate (Mar. 24).

Fowl Pest

Beds. 171, Castle Hill Road, Tottenhoe, Dunstable (Mar. 23); 173, Castle Hill Road, Tottenhoe, Dunstable (Mar. 24).

Cambs. Seven Stars Inn, The Avenue, March (Mar. 23).

Hants. Exbury Estates, Exbury, Southampton (Mar. 22).

Kent. New Farm, Pluckley, Ashford (Mar. 20).

Sussex. Oaklands Park, Hassocks (Mar. 22).

Swine Fever

Ayr. Duskridge, Beith (Mar. 26).

Ches. Elmtree Farm, Elworth, Sandbach (Mar. 23); Avondale Poultry Farm, Cross Lane, Marple, Stockport (Mar. 24); Beech Farm, Longsight Lane, Stanley Road, Cheadle Hulme (Mar. 25); Southfield Piggeries, Sagars Road, Styal, Wilmslow (Mar. 26).

Essex. The Piggeries, South View Road, West Thurrock, Grays (Mar. 23); "Thorpe," Wakering Road, Southchurch, Southend-on-Sea (Mar. 25); Scarletts, Distillery Road, Colchester (Mar. 26); Bentley Manor Farm, Lt. Bentley, Colchester; Modern Rays, Willows Green, Chelmsford (Mar. 28).

Glamorgan. Piggery, Llandaff Weir, Allotments, Cardiff (Mar. 25).

Lancs. Oak House, Weaste Lane, Thelwall, Warrington; Piggery, Rear Lodge, Agecroft Road, Pendlebury (Mar. 24); Barton Kennels, Moss Lane, Davyhulme, Nr. Manchester (Mar. 25); 185, Fleetwood Road, Thornton, Fleetwood (Mar. 26).

Lincs. Manor Farm, Lissington (Mar. 24).

Montgomery. Cilthrew, Llansantffraid (Mar. 22).

Norfolk. Church Farm, Mautby, Gt. Yarmouth (Mar. 22); The Piggeries, Rope Walk, Salters Road, Kings Lynn (Mar. 24).

Northants. Badby House, Badby, Daventry (Mar. 25).

Oxford. Ashwell House, Bloxham, Banbury (Mar. 22).

Renfrew. No. 8 Holding, East Fulton, Linwood, Paisley (Mar. 24).

Salop. Wood Lane Farm, Ellesmere (Mar. 22).

Staffs. Lymes Farm, Seabridge, Newcastle (Mar. 24); 2, Wolverhampton Road, Willenhall (Mar. 25); 90, Sandwell Road, West Bromwich; Rear of No. 24, Cobden Street, Walsall (Mar. 28).

Suffolk. Rose View, Saxon Street, Newmarket (Mar. 24).

Surrey. Vine Farm, Pirbright Road, Normandy, Guildford (Mar. 24).

Warwicks. Woodlands Farm, Hodgetts Lane, Berkswell, Coventry (Mar. 28).

Worcs. Piggeries, Forge Mill Lane, Redditch; Glebe Farm, Inkberrow (Mar. 24); 7, Arthur Street, Redditch (Mar. 26); Fairfield, Chapel Lane, South Littleton, Evesham (Mar. 28).

Yorks. Markenfield Hall, Nr. Ripon (Mar. 28).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

THE DISEASES OF SHEEP

Sir,—As Chairman of the Technical Development Committee, may I say how much I appreciate the receipt of the criticisms of the recent Revised Sheep Handbook made by Messrs. Marr & Sharman.

The work of the Technical Development Committee in the preparation of handbooks is necessarily long term, and from time to time the risk must arise that a contribution becomes some months out of date, especially if it has to be prepared at a time of rapid expansion of knowledge, more particularly in the clinical field. Every effort is made to avoid such dating being manifested too soon.

The criticisms received will be referred to the Committee for deliberation and appropriate action.

May I say that I am sure the members of the committee welcome constructive criticism which is indicative of the active interest of members and use of the Handbook, and which will act, I am sure, as a stimulus to future efforts.

Yours faithfully,

GEO. N. GOULD,

12, Landguard Road,
Southampton.

March 25th, 1955.

VETERINARY SURGEON EMERITUS

Sir,—I trust that the Council of the Royal College will ignore the letters of Dr. Hare and Mr. Gibson, and without any more hesitation remove the names of those members who have not paid their fees. If there are any of them so financially embarassed that they cannot afford the very low sum of one guinea the Veterinary Benevolent Fund might assist them. Some of these gentlemen have been in the Colonial Service and are on pension, but what is one guinea per year if they are still proud to be called veterinary surgeons? Let them be removed from the Register.

I should like our Editor to give us a list for comparison of what other professions are paying.

If the Council thought it advisable they might accept 10s. 6d. for those who have been qualified over 50 years; but in my opinion if they cannot afford one guinea they cannot afford 10s. 6d.

I consider the non-payment of these fees a sign of having lost all interest in life and especially in the profession.

Yours faithfully,

G. C. LANCASTER,

Eastwick House,
Evesham,
Worcester.

March 23rd, 1955.

We are informed that after payments respectively of five guineas and six guineas there are no annual fees for members of the medical profession. Chartered accountants pay annual fees ranging between four guineas and ten guineas. Solicitors pay £5 for admission and a practising fee of £3 per year.—EDITOR.

Sir,—Volume 67, page 208 of THE RECORD, containing a list of those gentlemen whose names had recently been removed from the Register, will always be thought of as a black page in our history by those of us who have a feeling for brethren in distress and for the comradeship of yesteryear.

May I urge the creation of a fund for the aid of those who, in difficult straits, would never appeal to others, but who would wish to retain their proper pride. The Register will be the richer by the retention of their names.

Yours faithfully,

IVOR HUGHES,

50, High Street,
Coleshill.

March 28th, 1955.

Our correspondent enclosed a donation with his letter, and this, with his consent, has been forwarded to the Victoria Veterinary Benevolent Fund.—EDITOR.

We much regret that owing to extreme pressure on our space several letters have been held over until next week.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 28th February, 1955		33	—	—	29	—	53
Corresponding period in	1954	8	—	5	83	—	54
	1953	33	—	1	11	—	94
	1952	12	—	4	6	1	48
1st January to 28th February, 1955 ...		184	—	5	140	—	193
Corresponding period in	1954	44	—	8	319	—	224
	1953	207	—	19	157	—	293
	1952	74	—	26	45	1	162

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 28th February, 1955, was as follows :—

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
69,369	32,448	33,592	135,409